Artwork by Jeanne Kelly
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**Note:** Words in **bold** are defined in the glossary at the end of this booklet.
The immune system is a network of **cells**, **tissues**, and **organs** that work together to defend the body against attacks by “foreign” invaders. These are primarily **microbes**—tiny **organisms** such as **bacteria**, **parasites**, and **fungi** that can cause infections. **Viruses** also cause infections, but are too primitive to be classified as living organisms. The human body provides an ideal environment for many microbes. It is the immune system’s job to keep them out or, failing that, to seek out and destroy them.

When the immune system hits the wrong target, however, it can unleash a torrent of disorders, including allergic diseases, arthritis, and a form of diabetes. If the immune system is crippled, other kinds of diseases result.

The immune system is amazingly complex. It can recognize and remember millions of different enemies, and it can produce secretions (release of fluids) and cells to match up with and wipe out nearly all of them.

The secret to its success is an elaborate and dynamic communications network. Millions and millions of cells, organized into sets and subsets, gather like clouds of bees swarming around a hive and pass information back and forth in response to an infection. Once immune cells receive the alarm, they become activated and begin to produce powerful chemicals. These substances allow the cells to regulate their own growth and behavior, enlist other immune cells, and direct the new recruits to trouble spots.
The key to a healthy immune system is its remarkable ability to distinguish between the body’s own cells, recognized as “self,” and foreign cells, or “nonself.” The body’s immune defenses normally coexist peacefully with cells that carry distinctive “self” marker molecules. But when immune defenders encounter foreign cells or organisms carrying markers that say “nonself,” they quickly launch an attack.

Anything that can trigger this immune response is called an antigen. An antigen can be a microbe such as a virus, or a part of a microbe such as a molecule. Tissues or cells from another person (except an identical twin) also carry nonself markers and act as foreign antigens. This explains why tissue transplants may be rejected.

Antigens carry marker molecules that identify them as foreign.
In abnormal situations, the immune system can mistake self for nonself and launch an attack against the body’s own cells or tissues. The result is called an **autoimmune disease**. Some forms of arthritis and diabetes are autoimmune diseases. In other cases, the immune system responds to a seemingly harmless foreign substance such as ragweed pollen. The result is allergy, and this kind of antigen is called an **allergen**.

**The Structure of the Immune System**

The organs of the immune system are positioned throughout the body. They are called **lymphoid organs** because they are home to **lymphocytes**, small white blood cells that are the key players in the immune system. Bone marrow, the soft tissue in the hollow center of bones, is the ultimate source of all blood cells, including lymphocytes. The thymus is a lymphoid organ that lies behind the breastbone. Lymphocytes known as **T lymphocytes** or **T cells** (“T” stands for “thymus”) mature in the thymus and then migrate to other tissues. **B lymphocytes**, also known as **B cells**, become activated and mature into **plasma cells**, which make and release **antibodies**.
The organs of the immune system are positioned throughout the body.
The lymph node contains numerous specialized structures. T cells concentrate in the paracortex, B cells in and around the germinal centers, and plasma cells in the medulla.

**Lymph nodes**, which are located in many parts of the body, are lymphoid tissues that contain numerous specialized structures.

- T cells from the thymus concentrate in the paracortex.
- B cells develop in and around the germinal centers.
- Plasma cells occur in the medulla.

Lymphocytes can travel throughout the body using the **blood vessels**. The cells can also travel through a system of **lymphatic vessels** that closely parallels the body’s **veins** and **arteries**.
Immune cells and foreign particles enter the lymph nodes via incoming lymphatic vessels or the lymph nodes’ tiny blood vessels.

Cells and fluids are exchanged between blood and lymphatic vessels, enabling the lymphatic system to monitor the body for invading microbes. The lymphatic vessels carry lymph, a clear fluid that bathes the body’s tissues.

Small, bean-shaped lymph nodes are laced along the lymphatic vessels, with clusters in the neck, armpits, abdomen, and groin. Each lymph node contains specialized compartments where immune cells congregate, and where they can encounter antigens.

Immune cells, microbes, and foreign antigens enter the lymph nodes via incoming lymphatic vessels or the lymph nodes’ tiny blood vessels. All lymphocytes exit lymph nodes through outgoing lymphatic vessels. Once in the bloodstream, lymphocytes are transported to tissues throughout the body. They patrol
everywhere for foreign antigens, then gradually drift back into the lymphatic system to begin the cycle all over again.

The spleen is a flattened organ at the upper left of the abdomen. Like the lymph nodes, the spleen contains specialized compartments where immune cells gather and work. The spleen serves as a meeting ground where immune defenses confront antigens.

Other clumps of lymphoid tissue are found in many parts of the body, especially in the linings of the digestive tract, airways, and lungs—territories that serve as gateways to the body. These tissues include the tonsils, adenoids, and appendix.

Immune Cells and Their Products

The immune system stockpiles a huge arsenal of cells, not only lymphocytes but also cell-devouring phagocytes and their relatives. Some immune cells take on all intruders, whereas others are trained on highly specific targets. To work effectively, most immune cells need the cooperation of their comrades. Sometimes immune cells communicate by direct physical contact, and sometimes they communicate releasing chemical messengers.

The immune system stores just a few of each kind of the different cells needed to recognize millions of possible enemies. When an antigen first appears, the few immune cells that can
respond to it multiply into a full-scale army of cells. After their job is done, the immune cells fade away, leaving sentries behind to watch for future attacks.

All immune cells begin as immature stem cells in the bone marrow. They respond to different cytokines and other chemical signals to grow into specific immune cell types, such as T cells, B cells, or phagocytes. Because stem cells have not yet committed to a particular future, their use presents an interesting possibility for treating some immune system disorders. Researchers currently are investigating if a person’s own stem cells can be used to regenerate damaged immune responses in autoimmune diseases and in immune deficiency disorders, such as HIV infection.

**B Cells**

B cells and T cells are the main types of lymphocytes. B cells work chiefly by secreting substances called antibodies into the body’s fluids. Antibodies ambush foreign antigens circulating in the bloodstream. They are powerless, however, to penetrate cells. The job of attacking target cells—either cells that have been infected by viruses or cells that have been distorted by cancer—is left to T cells or other immune cells (described below).

Each B cell is programmed to make one specific antibody. For example, one B cell will make an antibody that blocks a virus that causes the common cold, while another produces an antibody that attacks a bacterium that causes
B cells mature into plasma cells that produce antibodies.

pneumonia. When a B cell encounters the kind of antigen that triggers it to become active, it gives rise to many large cells known as plasma cells, which produce antibodies.

- **Immunoglobulin G**, or IgG, is a kind of antibody that works efficiently to coat microbes, speeding their uptake by other cells in the immune system.
- IgM is very effective at killing bacteria.
- IgA concentrates in body fluids—tears, saliva, and the secretions of the respiratory and digestive tracts—guarding the entrances to the body.
- IgE, whose natural job probably is to protect against parasitic infections, is responsible for the symptoms of allergy.
• IgD remains attached to B cells and plays a key role in initiating early B cell responses.

**T Cells**

Unlike B cells, T cells do not recognize free-floating antigens. Rather, their surfaces contain specialized antibody-like receptors that see fragments of antigens on the surfaces of infected or cancerous cells. T cells contribute to immune defenses in two major ways: some direct and regulate immune responses, whereas others directly attack infected or cancerous cells.

**Helper T cells,** or **Th cells,** coordinate immune responses by communicating with other cells. Some stimulate nearby B cells to produce antibodies, others call in microbe-gobbling cells called phagocytes, and still others activate other T cells.

**Cytotoxic T lymphocytes (CTLs)**—also called killer T cells—perform a different function. These cells directly attack other cells carrying certain foreign or abnormal molecules on their surfaces. CTLs are especially useful for attacking viruses because viruses often hide from other parts of the immune system while they grow inside infected cells. CTLs recognize small fragments of these viruses peeking out from the cell membrane and launch an attack to kill the infected cell.

In most cases, T cells only recognize an antigen if it is carried on the surface of a cell by one of the body’s own **major histocompatibility complex,** or MHC,
molecules. MHC molecules are proteins recognized by T cells when they distinguish between self and nonself. A self-MHC molecule provides a recognizable scaffolding to present a foreign antigen to the T cell. In humans, MHC antigens are called human leukocyte antigens, or HLA.

Although MHC molecules are required for T cell responses against foreign invaders, they also create problems during organ transplantations. Virtually every cell in the body is covered with MHC proteins, but each person has a different set of these proteins on his or her cells. If a T cell recognizes a nonself-MHC molecule on another cell, it will destroy the cell. Therefore, doctors must match organ recipients with donors who have the closest MHC makeup. Otherwise the recipient’s T cells will likely attack the transplanted organ, leading to graft rejection.

Some T cells are helper cells; others are killer cells.
Killer cell makes contact with target cell, trains its weapons on the target, then strikes.

**Natural killer (NK) cells** are another kind of lethal white cell, or lymphocyte. Like CTLs, NK cells are armed with granules filled with potent chemicals. But CTLs look for antigen fragments bound to self-MHC molecules, whereas NK cells recognize cells lacking self-MHC molecules. Thus, NK cells have the potential to attack many types of foreign cells.

Both kinds of killer cells slay on contact. The deadly assassins bind to their targets, aim their weapons, and then deliver a lethal burst of chemicals.

T cells aid the normal processes of the immune system. If NK T cells fail to function properly, asthma, certain autoimmune diseases—including type 1 diabetes—or the
growth of cancers may result. NK T cells get their name because they are a kind of T lymphocyte that carries some of the surface proteins, called “markers,” typical of NK T cells. But these T cells differ from other kinds of T cells. They do not recognize pieces of antigen bound to self-MHC molecules. Instead, they recognize fatty substances (lipids and glycolipids) that are bound to a different class of molecules called CD1d. Scientists are trying to discover methods to control the timing and release of chemical factors by NK T cells, with the hope they can modify immune responses in ways that benefit patients.

**Phagocytes and Their Relatives**

Phagocytes are large white cells that can swallow and digest microbes and other foreign particles. **Monocytes** are phagocytes that circulate in the blood. When monocytes migrate into tissues, they develop into **macrophages**. Specialized types of macrophages can be found in many organs, including the lungs, kidneys, brain, and liver.

Macrophages play many roles. As scavengers, they rid the body of worn-out cells and other debris. They display bits of foreign antigen in a way that draws the attention of matching lymphocytes and, in that respect, resemble **dendritic cells** (see page 15). And they churn out an amazing variety of powerful chemical signals, known as **monokines**, which are vital to the immune response.

**Granulocytes** are another kind of immune cell. They contain granules filled with potent chemicals, which allow the granulocytes to
Phagocytes, granulocytes, and mast cells, all with different methods of attack, demonstrate the immune system’s versatility.

destroy microorganisms. Some of these chemicals, such as histamine, also contribute to inflammation and allergy.

One type of granulocyte, the neutrophil, is also a phagocyte. Neutrophils use their prepackaged chemicals to break down the microbes they ingest. Eosinophils and basophils are granulocytes that “degranulate” by spraying their chemicals onto harmful cells or microbes nearby.

Mast cells function much like basophils, except they are not blood cells. Rather, they are found in the lungs, skin, tongue, and linings of the nose and intestinal tract, where they contribute to the symptoms of allergy.
Related structures, called blood **platelets**, are cell fragments. Platelets also contain granules. In addition to promoting blood clotting and wound repair, platelets activate some immune defenses.

Dendritic cells are found in the parts of lymphoid organs where T cells also exist. Like macrophages, dendritic cells in lymphoid tissues display antigens to T cells and help stimulate T cells during an immune response. They are called dendritic cells because they have branchlike extensions that can interlace to form a network.

**T Cell Receptors**

**T cell receptors** are complex protein molecules that peek through the surface membranes of T cells. The exterior part of a T cell receptor recognizes short pieces of foreign antigens that are bound to self-MHC molecules on other cells of the body. It is because of their T cell receptors that T cells can recognize disease-causing microorganisms and rally other immune cells to attack the invaders, or kill the invaders themselves.

**Toll-like receptors (TLRs)**, which occur on cells throughout the immune system, are a family of proteins the body uses as a first line of defense against invading microbes. Like T cell receptors, some TLRs peek through the surface membranes of immune cells, allowing them to respond to microbes in the cells’ environment.

Some TLRs are activated by molecules that make up viruses, whereas other TLRs respond to molecules that make up the cell walls of
bacteria. Once activated, TLRs relay the alarm to other actors in the immune system. For example, some TLRs play important roles in the all-purpose “first-responder” arm of the immune system, also called the innate immune system. In short order, the innate immune system responds with a surge of chemical signals that together cause inflammation, fever, and other responses to infection or injury. Other TLRs help initiate responses from genetically identical groups of lymphocytes, called clones, that are already programmed to recognize specific antigens. Such responses are called adaptive immunity.

Overall, the cellular receptors important for the first-line responses of innate immunity are encoded by genes people inherit from their parents. In contrast, adaptive immune responses rely on antigen receptors that are pieced together in the genomes of lymphocytes during their development in various tissues of the body. In addition to TLRs, other kinds of innate immune receptors can stimulate phagocytosis by macrophages, trigger the inflammatory responses that help control local infections, and play a range of crucial roles in defending the body against invading microbes.

**Cytokines**

Cells of the immune system communicate with one another by releasing and responding to chemical messengers called cytokines. These proteins are secreted by immune cells and act on other cells to coordinate appropriate immune responses. Cytokines include a diverse assortment of interleukins, interferons, and growth factors.
Cytokines include lymphokines, produced by lymphocytes, and monokines, made by monocytes and macrophages.

Some cytokines are chemical switches that turn certain immune cell types on and off. One cytokine, interleukin 2 (IL-2), triggers the immune system to produce T cells. IL-2’s immunity-boosting properties have traditionally made it a promising treatment for several illnesses. Clinical studies are underway to test its benefits in diseases such as cancer, hepatitis C, and HIV infection and AIDS. Scientists are studying other cytokines to see whether they can also be used to treat diseases.

One group of cytokines chemically attracts specific cell types. These so-called chemokines are released by cells at a site of injury or infection and call other immune cells to the region to help repair the damage or fight off the invader. Chemokines often play a key role in inflammation and are a promising target for new drugs to help regulate immune responses.
The complement system is made up of about 25 proteins that work together to assist, or “complement,” the action of antibodies in destroying bacteria. Complement also helps to rid the body of antibody-coated antigens (antigen-antibody complexes). Complement

The interlocking steps of the complement cascade end in cell death.
proteins, which cause blood vessels to become dilated and then leaky, contribute to the redness, warmth, swelling, pain, and loss of function that characterize an inflammatory response.

Complement proteins circulate in the blood in an inactive form. When the first protein in the complement series is activated—typically by antibody that has locked onto an antigen—it sets in motion a domino effect. Each component takes its turn in a precise chain of steps known as the complement cascade. The end products are molecular cylinders that are inserted into—and that puncture holes in—the cell walls that surround the invading bacteria. With fluids and molecules flowing in and out, the bacterial cells swell, burst, and die. Other components of the complement system make bacteria more susceptible to phagocytosis or beckon other immune cells to the area.

**Mounting an Immune Response**

Infections are the most common cause of human disease. They range from the common cold to debilitating conditions like chronic hepatitis to life-threatening diseases such as AIDS. Disease-causing microbes (pathogens) attempting to get into the body must first move past the body’s external armor, usually the skin or cells lining the body’s internal passageways.

The skin provides an imposing barrier to invading microbes. It is generally penetrable only through cuts or tiny abrasions. The digestive and respiratory tracts—both portals of entry for a number of microbes—also have
When challenged by a virus or other microbe, the immune system has many weapons to choose.

their own levels of protection. Microbes entering the nose often cause the nasal surfaces to secrete more protective mucus, and attempts to enter the nose or lungs can trigger a sneeze or cough reflex to force microbial invaders out of the respiratory passageways. The stomach contains a strong acid that destroys many pathogens that are swallowed with food.

If microbes survive the body’s front-line defenses, they still have to find a way through the walls of the digestive, respiratory, or urogenital passageways to the underlying cells. These passageways are lined with tightly packed epithelial cells covered in a layer of mucus, effectively blocking the transport of many pathogens into deeper cell layers.
Mucosal surfaces also secrete a special class of antibody called IgA, which in many cases is the first type of antibody to encounter an invading microbe. Underneath the epithelial layer a variety of immune cells, including macrophages, B cells, and T cells, lie in wait for any microbe that might bypass the barriers at the surface.

Next, invaders must escape a series of general defenses of the innate immune system, which are ready to attack without regard for specific antigen markers. These include patrolling phagocytes, NK T cells, and complement.

Microbes cross the general barriers then confront specific weapons of the adaptive immune system tailored just for them. These specific weapons, which include both antibodies and T cells, are equipped with singular receptor structures that allow them to recognize and interact with their designated targets.

**Bacteria, Viruses, and Parasites**

The most common disease-causing microbes are bacteria, viruses, and parasites. Each uses a different tactic to infect a person, and, therefore, each is thwarted by different components of the immune system.

Most bacteria live in the spaces between cells and are readily attacked by antibodies. When antibodies attach to a bacterium, they send signals to complement proteins and phagocytic cells to destroy the bound microbes. Some bacteria are eaten directly by phagocytes, which signal to certain T cells to join the attack.
The combination of antigen fragment and MHC molecule attracts the help of a mature, matching T cell.

Antibodies are triggered when a B cell encounters its matching antigen.

The B cell takes in the antigen and digests it,

then it displays antigen fragments bound to its own distinctive MHC molecules.

The combination of antigen fragment and MHC molecule attracts the help of a mature, matching T cell.

Lymphokines secreted by the T cell allow the B cell to multiply and mature into antibody-producing plasma cells.

Released into the bloodstream, antibodies lock onto matching antigens. These antigen-antibody complexes are soon eliminated, either by the complement cascade or by the liver and the spleen.

B cells are triggered to mature into plasma cells that produce a specific kind of antibody when the B cell encounters a specific antigen.
T cells are mobilized when they encounter a cell such as a macrophage or a B cell that has digested an antigen and is displaying antigen fragments bound to its MHC molecules. Lymphokines help the T cell to mature. The T cell, alerted and activated, secretes lymphokines.

Some lymphokines attract immune cells—fresh macrophages, granulocytes, and other lymphocytes—to the site of infection. Yet other lymphokines direct the recruits once they arrive on the scene. Some T cells become killer cells and track down body cells infected by viruses. Some lymphokines spur the growth of more T cells.

T cells become active through a series of steps and then activate other immune cells by secreting lymphokines.
All viruses, plus a few types of bacteria and parasites, must enter cells of the body to survive, requiring a different kind of immune defense. Infected cells use their MHC molecules to put pieces of the invading microbes on their surfaces, flagging down CTLs to destroy the infected cells. Antibodies also can assist in the immune response by attaching to and clearing viruses before they have a chance to enter cells.

Parasites live either inside or outside cells. Intracellular parasites such as the organism that causes malaria can trigger T cell responses. Extracellular parasites are often much larger than bacteria or viruses and require a much broader immune attack. Parasitic infections often trigger an inflammatory response in which eosinophils, basophils, and other specialized granule-containing cells rush to the scene and release their stores of toxic chemicals in an attempt to destroy the invaders. Antibodies also play a role in this attack, attracting the granule-filled cells to the site of infection.
Immunity: Natural and Acquired

Long ago, physicians realized that people who had recovered from the plague would never get it again—they had acquired immunity. This is because some of the activated T and B cells had become memory cells. Memory cells ensure that the next time a person meets up with the same antigen, the immune system is already set to demolish it.

Immunity can be strong or weak, short-lived or long-lasting, depending on the type of antigen it encounters, the amount of antigen, and the route by which the antigen enters the body. Immunity can also be influenced by inherited genes. When faced with the same antigen, some individuals will respond forcefully, others feebly, and some not at all.
An immune response can be sparked not only by infection but also by immunization with vaccines. Some vaccines contain microorganisms—or parts of microorganisms—that have been treated so they can provoke an immune response but not full-blown disease. (See “Vaccines” on page 27.)

Immunity can also be transferred from one individual to another by injections of serum rich in antibodies against a particular microbe (antiserum). For example, antiserum is sometimes given to protect travelers to countries where hepatitis A is widespread. The antiserum induces passive immunity against the hepatitis A virus. Passive immunity typically lasts only a few weeks or months.

Infants are born with weak immune responses but are protected for the first few months of life by antibodies they receive from their mothers before birth. Babies who are nursed can also receive some antibodies from breast milk that help to protect their digestive tracts.

**Immune Tolerance**

Immune tolerance is the tendency of T or B lymphocytes to ignore the body’s own tissues. Maintaining tolerance is important because it prevents the immune system from attacking its fellow cells. Scientists are hard at work trying to understand how the immune system knows when to respond and when to ignore an antigen.
Tolerance occurs in at least two ways—central tolerance and peripheral tolerance. Central tolerance occurs during lymphocyte development. Very early in each immune cell’s life, it is exposed to many of the self molecules in the body. If it encounters these molecules before it has fully matured, the encounter activates an internal self-destruct pathway, and the immune cell dies. This process, called clonal deletion, helps ensure that “self-reactive” T cells and B cells, those that could develop the ability to destroy the body’s own cells, do not mature and attack healthy tissues.

Because maturing lymphocytes do not encounter every molecule in the body, they must also learn to ignore mature cells and tissues. In peripheral tolerance, circulating lymphocytes might recognize a self molecule but cannot respond because some of the chemical signals required to activate the T or B cell are absent. So-called clonal anergy, therefore, keeps potentially harmful lymphocytes switched off. Peripheral tolerance may also be imposed by a special class of regulatory T cells that inhibits helper or cytotoxic T-cell activation by self antigens.

Vaccines
For many years, healthcare providers have used vaccination to help the body’s immune system prepare for future attacks. Vaccines consist of killed or modified microbes, components of microbes, or microbial DNA that trick the body into thinking an infection has occurred.
A vaccinated person’s immune system attacks the harmless vaccine and prepares for invasions against the kind of microbe the vaccine contained. In this way, the person becomes immunized against the microbe. Vaccination remains one of the best ways to prevent infectious diseases, and vaccines have an excellent safety record. Previously devastating diseases such as smallpox, polio, and whooping cough have been greatly controlled or eliminated through worldwide vaccination programs.

Disorders of the Immune System

Allergic Diseases
The most common types of allergic diseases occur when the immune system responds to a false alarm. In an allergic person, a normally harmless material such as grass pollen, food particles, mold, or house dust mites is mistaken for a threat and attacked.

Allergies such as pollen allergy are related to the antibody known as IgE. Like other antibodies, each IgE antibody is specific; one acts against oak pollen and another against ragweed, for example.

Autoimmune Diseases
Sometimes the immune system’s recognition apparatus breaks down, and the body begins to manufacture T cells and antibodies directed against self antigens in its own cells and tissues. As a result, healthy cells and tissues
The first time the allergy-prone person runs across an allergen such as ragweed, he or she makes large amounts of ragweed IgE antibody. These IgE molecules attach themselves to mast cells.

The second time that person has a brush with ragweed, the IgE-primed mast cell will release its powerful chemicals, and the person will suffer the wheezing and/or sneezing, runny nose, watery eyes, and itching of allergy.

An allergic reaction occurs after plasma cells produce IgE antibody against a specific antigen and mast cells become activated.
Misguided T cells can attack insulin-producing cells of the pancreas, contributing to an autoimmune form of diabetes.

are destroyed, which leaves the person’s body unable to perform important functions.

Misguided T cells and autoantibodies, as they are known, contribute to many autoimmune diseases. For instance, T cells that attack certain kinds of cells in the pancreas contribute to a form of diabetes, whereas an autoantibody known as rheumatoid factor is common in people with rheumatoid arthritis. People with systemic lupus erythematosus (SLE) have antibodies to many types of their own cells and cell components. SLE patients can develop a severe rash, serious kidney inflammation, and disorders of other important tissues and organs.

No one knows exactly what causes an autoimmune disease, but multiple factors are likely to be involved. These include elements in the environment, such as viruses, certain drugs, and sunlight, all of which may damage
or alter normal body cells. Hormones are suspected of playing a role because most autoimmune diseases are far more common in women than in men. Heredity, too, seems to be important. Many people with autoimmune diseases have characteristic types of self-marker molecules.

**Immune Complex Diseases**

Immune complexes are clusters of interlocking antigens and antibodies. Normally, immune complexes are rapidly removed from the bloodstream. Sometimes, however, they continue to circulate and eventually become trapped in the tissues of the kidneys, lungs, skin, joints, or blood vessels. There, they set off reactions with complement that lead to

Antigen-antibody complexes can become trapped in, and damage, the kidneys and other organs.
inflammation and tissue damage. Immune complexes work their mischief in many diseases. These include malaria and viral hepatitis, as well as many autoimmune diseases.

**Immune Deficiency Disorders**

When the immune system is missing one or more of its parts, the result is an immune deficiency disorder. These disorders can be inherited, acquired through infection, or produced as a side effect by drugs such as those used to treat people with cancer or those who have received transplants.

Temporary immune deficiencies can develop in the wake of common virus infections, including influenza, infectious mononucleosis, and measles. Immune responses can also be depressed by blood transfusions, surgery, malnutrition, smoking, and stress.

Some children are born with poorly functioning immune systems. Some have flaws in the B cell system and cannot produce antibodies. Others, whose thymus is either missing or small and abnormal, lack T cells. Very rarely, infants are born lacking all of the major immune defenses. This condition is known as severe combined immune deficiency disease or SCID. (See “Gene Therapy” on page 39.)
The AIDS virus takes over the machinery of the T cells it infects, using the cells to make new viruses.

AIDS is an immune deficiency disorder caused by a virus (HIV) that infects immune cells. HIV can destroy or disable vital T cells, paving the way for a variety of immunologic shortcomings. The virus also can hide out for long periods in immune cells. As the immune defenses falter, a person develops AIDS and falls prey to unusual, often life-threatening infections and rare cancers.
Each year thousands of lives in the United States are prolonged by transplanted organs including the kidneys, heart, lung, liver, and pancreas. For a transplant to “take,” however, the body’s natural tendency to rid itself of foreign tissue must be overridden.

One way to avoid the rejection of transplanted tissue is tissue typing, which ensures that markers of self on the donor’s tissue are as similar as possible to those of the recipient. Every cell in the body has a double set of six major tissue antigens, and each of the antigens exists, in different individuals, in as many as 20 varieties. The chance of two people having identical transplant antigens is about one in 100,000.

A second way to avoid transplant rejection is to lull the recipient’s immune system into a less active state. This can be done with powerful immunosuppressive drugs such as cyclosporine A, or by using laboratory-manufactured antibodies that attack mature T cells.

**Bone Marrow Transplants**

When the immune response is severely depressed—in infants born with immune disorders or in people with cancer, for example—one possible remedy is a transfer of healthy bone marrow. Once introduced into the circulation, transplanted bone marrow cells can develop into functioning B and T cells.
In bone marrow transplants, a close match is extremely important. Not only is there a danger that the body will reject the transplanted bone marrow cells, but mature T cells from the bone marrow transplant may counterattack and destroy the recipient’s tissues. To prevent this situation, known as **graft-versus-host disease**, scientists use drugs or antibodies to “cleanse” the donor marrow of potentially dangerous mature T cells.

The Immune System and the Nervous System

Evidence is mounting that the immune system and the nervous system are linked in several ways. One well-known connection involves the **adrenal glands**. In response to stress messages from the brain, the adrenal glands release hormones into the blood. In addition to helping a person respond to emergencies by mobilizing the body’s energy reserves, these “stress hormones” can stifle the protective effects of antibodies and lymphocytes.

Another link between the immune system and the nervous system is that the hormones and other chemicals that convey messages among nerve cells also “speak” to cells of the immune system. Indeed, some immune cells are able to manufacture typical nerve cell products, and some **lymphokines** can transmit information to the nervous system. Moreover, the brain may send messages directly down nerve cells to the immune system. Networks of nerve fibers have been found connecting to the lymphoid organs.
Scientists are now able to mass-produce immune cell secretions, both antibodies and lymphokines, as well as specialized immune cells. The ready supply of these materials not only has revolutionized the study of the immune system itself but also has had an enormous impact on medicine, agriculture, and industry.

**Monoclonal antibodies** are identical antibodies made by the many clones of a single B cell.
Because of their unique specificity for different antigens, monoclonal antibodies are promising treatments for a range of diseases. Researchers make monoclonal antibodies by injecting a mouse with a target antigen and then fusing B cells from the mouse with other long-lived cells. The resulting hybrid cell becomes a type of antibody factory, turning out identical copies of antibody molecules specific for the target antigen.

Mouse antibodies are “foreign” to people, however, and might trigger an immune response when injected into a human. Therefore, researchers have developed “humanized” monoclonal antibodies. To construct these molecules, scientists take the antigen-binding portion of a mouse antibody and attach it to a human antibody scaffolding, greatly reducing the foreign portion of the molecule.

Because they recognize very specific molecules, monoclonal antibodies are used in diagnostic tests to identify invading pathogens or changes in the body’s proteins. In medicine, monoclonal antibodies can attach to cancer cells, blocking the chemical growth signals that cause the cells to divide out of control. In other cases, monoclonal antibodies can carry potent toxins into certain cells, killing the dangerous cells while leaving their neighbors untouched.

**Genetic Engineering**

Genetic engineering allows scientists to pluck genes—segments of DNA—from one type of organism and combine them with genes of a second organism. In this way, relatively simple
Cytokine-producing cell

Strand of DNA from cytokine-producing cell

Cytokine gene is cut out of DNA

Plasmid—a ring of DNA—from bacterium

Plasmid is cut open

Cytokine gene is spliced into plasmid

Hybrid plasmid is put back into bacterium

Bacterium makes human cytokines

Genetic engineering transforms simple organisms into factories for making human proteins.
organisms such as bacteria or yeast (a type of fungus) can be induced to make quantities of human proteins, including hormones such as insulin as well as lymphokines and monokines. They can also manufacture proteins from infectious agents, such as the hepatitis virus or HIV, for use in vaccines.

**Gene Therapy**

Genetic engineering also holds promise for gene therapy—replacing altered or missing genes or adding helpful genes. One disease in which gene therapy has been successful is SCID, or severe combined immune deficiency disease.

SCID is a rare genetic disease that disables a person’s immune system and leaves the person unable to fight off infections. It is caused by mutations in one of several genes that code for important components of the immune system. Until recently, the most effective treatment for SCID was transplantation of blood-forming stem cells from the bone marrow of a healthy person who is closely related to the patient. However, doctors have also been able to treat SCID by giving the patient a genetically engineered version of the missing gene.

Using gene therapy to treat SCID is generally accomplished by taking blood-forming cells from a person’s own bone marrow, introducing into the cells a genetically changed virus that carries the corrective gene, and growing the modified cells outside the person’s body. After the genetically changed bone marrow cells
begin to produce the **enzyme** or other protein that was missing, the modified blood-forming marrow cells can be injected back into the person. Once back inside the body, the genetically modified cells can produce the missing immune system component and begin to restore the person’s ability to fight off infections.

Cancer is another target for gene therapy. In pioneering experiments, scientists are removing cancer-fighting lymphocytes from the cancer patient’s tumor, inserting a gene that boosts the lymphocytes’ ability to make quantities of a natural anticancer product, then growing the restructured cells in quantity in the laboratory. These cells are injected back into the person, where they can seek out the tumor and deliver large doses of the anticancer chemical.

**Immunoregulation**

Research into the delicate checks and balances that control the immune response is increasing knowledge of normal and abnormal immune system functions. Someday it may be possible to treat autoimmune diseases such as systemic lupus erythematosus by suppressing parts of the immune system that are overactive.
The SCID-hu mouse provides a means of studying the human immune system in action.

Scientists are also devising ways to better understand the human immune system and diseases that affect it. For example, by transplanting immature human immune tissues or immune cells into SCID mice, scientists have created “humanized” mice, a living model of the human immune system. Scientists are manipulating the immune system of humanized SCID mice to discover ways to benefit human health. Humanized mice are also being used in research on transplantation and autoimmune and allergic diseases, and to manufacture molecules that help regulate immune system function and immune tolerance.
Although scientists have learned much about the immune system, they continue to study how the body launches attacks that destroy invading microbes, infected cells, and tumors while ignoring healthy tissues. New technologies for identifying individual immune cells are now allowing scientists to determine quickly which targets are triggering an immune response. Improvements in microscopy are permitting the first-ever observations of living B cells, T cells, and other cells as they interact within lymph nodes and other body tissues.

In addition, scientists are rapidly unraveling the genetic blueprints that direct the human immune response, as well as those that dictate the biology of bacteria, viruses, and parasites. The combination of new technology and expanded genetic information will no doubt reveal even more about how the body protects itself from disease.
Glossary

**adenoids**—see tonsils.

**adrenal gland**—a gland located on each kidney that secretes hormones regulating metabolism, sexual function, water balance, and stress.

**allergen**—any substance that causes an allergy.

**antibody**—a molecule (also called an immunoglobulin) produced by a mature B cell (plasma cell) in response to an antigen. When an antibody attaches to an antigen, it helps the body destroy or inactivate the antigen.

**antigen**—a substance or molecule that is recognized by the immune system. The antigen can be from foreign material such as bacteria or viruses.

**antiserum**—a serum rich in antibodies against a particular microbe.

**appendix**—lymphoid organ in the intestine.

**artery**—a blood vessel that carries blood from the heart to other parts of the body.

**autoantibody**—an antibody that reacts against a person’s own tissue.

**autoimmune disease**—disease that results when the immune system mistakenly attacks the body’s own tissues. Examples include multiple sclerosis, type 1 diabetes, rheumatoid arthritis, and systemic lupus erythematosus.
**B cell or B lymphocyte**—a small white blood cell crucial to the immune defenses. B cells come from bone marrow and develop into blood cells called plasma cells, which are the source of antibodies.

**bacteria**—microscopic organisms composed of a single cell. Some cause disease.

**basophil**—a white blood cell that contributes to inflammatory reactions. Along with mast cells, basophils are responsible for the symptoms of allergy.

**blood vessel**—an artery, vein, or capillary that carries blood to and from the heart and body tissues.

**cell**—the smallest unit of life; the basic living unit that makes up tissues.

**chemokine**—a small protein molecule that activates immune cells, stimulates their migration, and helps direct immune cell traffic throughout the body.

**clonal anergy**—the process of switching off the ability of potentially harmful T or B cells to participate in immune responses. Clonal anergy is essential for generating the tolerance of T and B cells to the body’s “self” tissue antigens.
**Clonal deletion**—the genetically controlled process of eliminating immune cells that could destroy the body’s own cells and tissues. The elimination process removes immature T and B lymphocytes that have receptors for cells with “self” MHC or HLA antigens, and could therefore attack and destroy the body’s own cells.

**Clone**—a group of genetically identical cells or organisms descended from a single common ancestor; or, to reproduce identical copies.

**Complement**—a complex series of blood proteins whose action “complements” the work of antibodies. Complement destroys bacteria, produces inflammation, and regulates immune reactions.

**Complement cascade**—a precise sequence of events, usually triggered by antigen-antibody complexes, in which each component of the complement system is activated in turn.

**Cytokines**—powerful chemical substances secreted by cells that enable the body’s cells to communicate with one another. Cytokines include lymphokines produced by lymphocytes and monokines produced by monocytes and macrophages.

**Cytotoxic T lymphocyte (CTL)**—a subtype of T cells that carries the CD8 marker and can destroy body cells infected by viruses or transformed by cancer.
**dendritic cell**—an immune cell with highly branched extensions that occurs in lymphoid tissues, engulfs microbes, and stimulates T cells by displaying the foreign antigens of the microbes on their surfaces.

**DNA (deoxyribonucleic acid)**—a long molecule found in the cell nucleus. Molecules of DNA carry the cell’s genetic information.

**enzyme**—a protein produced by living cells that promotes the chemical processes of life without itself being altered.

**eosinophil**—a white blood cell containing granules filled with chemicals damaging to parasites and enzymes that affect inflammatory reactions.

**epithelial cells**—cells that make up the epithelium, the covering for internal and external body surfaces.

**fungus**—a member of a class of relatively primitive vegetable organisms. Fungi include mushrooms, yeasts, rusts, molds, and smuts.

**gene**—a unit of genetic material (DNA) inherited from a parent that controls specific characteristics. Genes carry coded directions a cell uses to make specific proteins that perform specific functions.

**genome**—a full set of genes in a person or any other living thing.
**graft rejection**—an immune response against transplanted tissue.

**graft-versus-host disease**—a life-threatening reaction in which transplanted cells attack the tissues of the recipient.

**granule**—a membrane-bound organelle (specialized part) within cells where proteins are stored before secretion.

**granulocyte**—a phagocytic white blood cell filled with granules. Neutrophils, eosinophils, basophils, and mast cells are examples of granulocytes.

**growth factors**—chemicals secreted by cells that stimulate proliferation of or changes in the physical properties of other cells.

**helper T cells (Th cells)**—a subset of T cells that carry the CD4 surface marker and are essential for turning on antibody production, activating cytotoxic T cells, and initiating many other immune functions.

**hepatitis**—the name of several viruses that cause liver diseases. These viruses include hepatitis A, hepatitis B, and hepatitis C.

**histocompatibility testing**—a test conducted before transplant operations to find a donor whose MHC molecules are similar to the recipient’s; helps reduce the strength of transplant rejection.
HIV (human immunodeficiency virus)—the virus that causes AIDS.

**human leukocyte antigen (HLA)**—a protein on the surfaces of human cells that identifies the cells as “self” and, like MHC antigens, performs essential roles in immune responses. HLAs are used in laboratory tests to determine whether one person’s tissues are compatible with another person’s, and could be used in a transplant. HLAs are the human equivalent of MHC antigens; they are coded for by MHC genes.

**immune response**—reaction of the immune system to foreign substances. Although normal immune responses are designed to protect the body from pathogens, immune dysregulation can damage normal cells and tissues, as in the case of autoimmune diseases.

**immunoglobulin**—one of a family of large protein molecules, also known as antibodies, produced by mature B cells (plasma cells).

**immunosuppressive**—capable of reducing immune responses.

**inflammation**—an immune system reaction to “foreign” invaders such as microbes or allergens. Signs include redness, swelling, pain, or heat.

**inflammatory response**—redness, warmth, and swelling produced in response to infection; the result of increased blood flow and an influx of immune cells and their secretions.
**innate**—an immune system function that is inborn and provides an all-purpose defense against invasion by microbes.

**interferon**—a protein produced by cells that stimulates antivirus immune responses or alters the physical properties of immune cells.

**interleukins**—a major group of lymphokines and monokines.

**lymph**—a transparent, slightly yellow fluid that carries lymphocytes, bathes the body tissues, and drains into the lymphatic vessels.

**lymph node**—a small bean-shaped organ of the immune system, distributed widely throughout the body and linked by lymphatic vessels. Lymph nodes are garrisons of B and T cells, dendritic cells, macrophages, and other kinds of immune cells.

**lymphatic vessels**—a bodywide network of channels, similar to the blood vessels, which transports lymph to the immune organs and into the bloodstream.

**lymphocyte**—a small white blood cell produced in the lymphoid organs and essential to immune defenses. B cells, T cells, and NK T cells are lymphocytes.
lymphoid organ—an organ of the immune system where lymphocytes develop and congregate. These organs include the bone marrow, thymus, lymph nodes, spleen, and various other clusters of lymphoid tissue. Blood vessels and lymphatic vessels are also lymphoid organs.

lymphokines—powerful chemical substances secreted by lymphocytes. These molecules help direct and regulate the immune responses.

macrophage—a large and versatile immune cell that devours invading pathogens and other intruders. Macrophages stimulate other immune cells by presenting them with small pieces of the invaders.

major histocompatibility complex (MHC)—a group of genes that controls several aspects of the immune response. MHC genes code for “self” markers on all body cells.

mast cell—a granulocyte found in tissue. The contents of mast cells, along with those of basophils, are responsible for the symptoms of allergy.

memory cells—a subset of T cells and B cells that have been exposed to antigens and can then respond more readily when the immune system encounters those same antigens again.
**microbe** or **microorganism**—a microscopic living organism. Examples include bacteria, protozoa, and some fungi and parasites. Viruses are also called microbes.

**molecule**—the smallest amount of a specific chemical substance. Large molecules such as proteins, fats, carbohydrates, and nucleic acids are the building blocks of a cell, and a gene determines how each molecule is produced.

**monoclonal antibody**—an antibody produced by a single B cell or its identical progeny that is specific for a given antigen. Monoclonal antibodies are used as research tools for binding to specific protein molecules, and are invaluable in research, medicine, and industry.

**monocyte**—a large phagocytic white blood cell which, when entering tissue, develops into a macrophage.

**monokines**—powerful chemical substances secreted by monocytes and macrophages. These molecules help direct and regulate the immune responses.

**natural killer (NK) cell**—a large granule-containing lymphocyte that recognizes and kills cells lacking self antigens. These cells’ target recognition molecules are different from T cells.

**NK T cell**—a T cell that has some characteristics of NK cells. It produces large amounts of cytokines when stimulated, and is activated by fatty substances (lipids) bound to non-MHC molecules called CD1d.
**neutrophil**—a white blood cell that is an abundant and important phagocyte.

**organ**—a part of the body that has a specific function, such as the lungs.

**organism**—an individual living thing composed of one or more cells.

**parasite**—a plant or animal that lives, grows, and feeds on or within another living organism.

**passive immunity**—immunity resulting from the transfer of antibodies or antiserum produced by another person.

**pathogen**—a disease-causing organism or virus.

**phagocyte**—a large white blood cell that contributes to immune defenses by ingesting microbes or other cells and foreign particles.

**phagocytosis**—process by which one cell engulfs another cell or large particle.

**plasma cell**—a large antibody-producing cell that develops from B cells.

**platelet**—a cellular fragment critical for blood clotting and sealing off wounds.

**serum**—the clear liquid that separates from the blood when it is allowed to clot. This fluid contains the antibodies that were present in the whole blood.
spleen—a lymphoid organ in the abdominal cavity that is an important center for immune system activities.

stem cell—an immature cell from which other cells derive. Bone marrow is rich in the kind of stem cells that become specialized blood cells.

T cell or T lymphocyte—a small white blood cell that recognizes antigen fragments bound to cell surfaces by specialized antibody-like receptors. “T” stands for the thymus gland, where T cells develop and acquire their receptors.

T cell receptor—complex protein molecule on the surfaces of T cells that recognizes bits of foreign antigen bound to self-MHC molecules.

Toll-like receptor (TLR)—a family of proteins important for first-line immune defenses against microbes.

tissue typing—see histocompatibility testing.

tissue—a group of similar cells joined to perform the same function.

tolerance—a state of immune nonresponsiveness to a particular antigen or group of antigens.

tonsils and adenoids—prominent oval masses of lymphoid tissues on either side of the throat.

toxin—an agent produced in plants and bacteria, normally very damaging to cells.
**vaccine**—a preparation that stimulates an immune response that can prevent an infection or create resistance to an infection. Vaccines do not cause disease.

**vein**—a blood vessel that carries blood to the heart from the body tissues.

**virus**—a particle composed of a piece of genetic material—RNA or DNA—surrounded by a protein coat. Viruses can reproduce only in living cells.
INTRODUCTION TO HIV AND AIDS

AS REQUIRED PER FLORIDA STATUTE 456.13(7)

This article is from the National Library of Medicine. It a chapter from the book published on the web by the National Library of Medicine. It has been compiled and abridged by Complete Counseling.

AHCPR Archived reports, Put Prevention Into Practice and Minnesota Health Technology Advisory Committee ➦ SAMHSA/CSAT Treatment Improvement Protocols ➦ 37. TIP 37: Substance Abuse Treatment for Persons with HIV/AIDS
Chapter 1-- Introduction to HIV/AIDS

The first cases of acquired immunodeficiency syndrome (AIDS) were reported in the United States in the spring of 1981. By 1983 the human immunodeficiency virus (HIV), the virus that causes AIDS, had been isolated. Early in the U.S. HIV/AIDS pandemic, the role of substance abuse in the spread of AIDS was clearly established. Injection drug use (IDU) was identified as a direct route of HIV infection and transmission among injection drug users. The largest group of early AIDS cases comprised gay and bisexual men (referred to as men who have sex with men or MSMs). Early cases of HIV infection that were sexually transmitted often were related to the use of alcohol and other substances, and the majority of these cases occurred in urban, educated, white MSMs.

Currently, injection drug users represent the largest HIV-infected substance-abusing population in the United States. HIV/AIDS prevalence rates among injection drug users vary by geographic region, with the highest rates in surveyed substance abuse treatment centers in the Northeast, the South, and Puerto Rico. From July 1998 through June 1999, 23 percent of all AIDS cases reported were among men and women who reported IDU (Centers for Disease Control and Prevention [CDC], 1999b).

IDU practices are quick and efficient vehicles for HIV transmission. The virus is transmitted primarily through the exchange of blood using needles, syringes, or other IDU equipment (e.g., cookers, rinse water, cotton) that were previously used by an HIV-infected person. Lack of knowledge about safer needle use techniques and the lack of alternatives to needle sharing (e.g., available supplies of clean, new needles) contribute to the rise of HIV/AIDS.

Another route of HIV transmission among injection drug users is through sexual contacts within relatively closed sexual networks, which are characterized by multiple sex partners, unprotected sexual intercourse, and exchange of sex for money (Friedman et al., 1995). The inclusion of alcohol and other noninjection substances to this lethal mixture only increases the HIV/AIDS caseload (Edlin et al., 1994; Grella et al., 1995). A major risk factor for HIV/AIDS among injection drug users is crack use; one study found that crack abusers reported more sexual partners in the last 12 months, more sexually transmitted diseases (STDs) in their lifetimes, and greater frequency of paying for sex, exchanging sex for drugs, and having sex with injection drug users (Word and Bowser, 1997).

Following are the key concepts about HIV/AIDS and substance abuse disorders that influenced the creation of this TIP:

- **Substance abuse increases the risk of contracting HIV.** HIV infection is substantially associated with the use of contaminated or used needles to inject heroin. Also, substance abusers may put themselves at risk for HIV infection by engaging in risky sex behaviors in exchange for powder or crack cocaine. However, this fact does not minimize the impact of other substances that may be used (e.g., hallucinogens, inhalants, stimulants, prescription medications).
- **Substance abusers are at risk for HIV infection through sexual behaviors.** Both men and women may engage in risky sexual behaviors (e.g., unprotected
anal, vaginal, or oral sex; sharing of sex toys; handling or consuming body fluids and body waste; sex with infected partners) for the purpose of obtaining substances, while under the influence of substances, or while under coercion.

- **Substance abuse treatment serves as HIV prevention.** Placing the client in substance abuse treatment along a continuum of care and treatment helps minimize continued risky substance-abusing practices. Reducing a client's involvement in substance-abusing practices reduces the probability of infection.

- **HIV/AIDS, substance abuse disorders, and mental disorders interact in a complex fashion.** Each acts as a potential catalyst or obstacle in the treatment of the other two—substance abuse can negatively affect adherence to HIV/AIDS treatment regimens; substance abuse disorders and HIV/AIDS are intertwining disorders; HIV/AIDS is changing the shape and face of substance abuse treatment; complex and legal issues arise when treating HIV/AIDS and substance abuse; HIV-infected women with substance abuse disorders have special needs.

- **Risk reduction allows for a comprehensive approach to HIV/AIDS prevention.** This strategy promotes changing substance-related and sex-related behaviors to reduce clients' risk of contracting or transmitting HIV.

The first part of this chapter provides a basic overview of the origin of HIV/AIDS and the transmission and progression of the disease. The second part of the chapter presents a summary of epidemiological data from the CDC. This second part discusses the impact of HIV/AIDS in regions of the United States and the populations that are at the greatest risk of contracting HIV.

**Overview of HIV/AIDS**

**Origin of HIV/AIDS**

Of the many theories and myths about the origin of HIV, the most likely explanation is that HIV was introduced to humans from monkeys. A recent study (Gao et al., 1999) identified a subspecies of chimpanzees native to west equatorial Africa as the original source of HIV-1, the virus responsible for the global AIDS pandemic. The researchers believe that the virus crossed over from monkeys to humans when hunters became exposed to infected blood. Monkeys can carry a virus similar to HIV, known as SIV (simian immunodeficiency virus), and there is strong evidence that HIV and SIV are closely related (Simon et al., 1998; Zhu et al., 1998).

AIDS is caused by HIV infection and is characterized by a severe reduction in CD4+ T cells, which means an infected person develops a very weak immune system and becomes vulnerable to contracting life-threatening infections (such as Pneumocystis carinii pneumonia). AIDS occurs late in HIV disease.

Tracking of the disease in the United States began early after the discovery of the pandemic, but even to date, tracking data reveal only how many individuals have AIDS, not how many have HIV. The counted AIDS cases are like the visible part of an iceberg, while the much larger portion, HIV, is submerged out of sight. Many States are counting HIV cases now that positive results are to be gained by treating the infection in the early stages and because counting only
AIDS cases is no longer sufficient for projecting trends of the pandemic. However, because HIV-infected people generally are asymptomatic for years, they might not be tested or included in the count. The CDC estimates that between 650,000 and 900,000 people in the United States currently are living with HIV (CDC, 1997c).

In 1996, the number of new AIDS cases (not HIV cases) and deaths from AIDS began to decline in the United States for the first time since 1981. Deaths from AIDS have decreased since 1996 in all racial and ethnic groups and among both men and women (CDC, 1999a). However, the most recent CDC data show that the decline is slowing (CDC, 1999b). The decline can be attributed to advances in treating HIV with multiple medications, known as combination therapy; treatments to prevent secondary opportunistic infections; and a reduction in the HIV infection rate in the mid-1980s prior to the introduction of combination therapy. The latter can be attributed to improved services for people with HIV and access to health care. In general, those with the best access to good, ongoing HIV/AIDS care increase their chances of living longer.

HIV/AIDS is still largely a disease of MSMs and male injection drug users, but it is spreading most rapidly among women and adolescents, particularly in African American and Hispanic communities. HIV is a virus that thrives in certain ecological conditions. The following will lead to higher infection rates: a more potent virus, high viral load, high prevalence of STDs, substance abuse, high HIV seroprevalence within the community, high rate of unprotected sexual contact with multiple partners, and low access to health care. These ecological conditions exist to a large degree among urban, poor, and marginalized communities of injection drug users. Thus, MSMs and African American and Hispanic women, their children, and adolescents within these communities are at greatest risk.

**HIV Transmission**

HIV cannot survive outside of a human cell. HIV must be transmitted directly from one person to another through human body fluids that contain HIV-infected cells, such as blood, semen, vaginal secretions, or breast milk. The most effective means of transmitting HIV is by direct contact between the infected blood of one person and the blood supply of another. (See Figure 1-1 for an illustration of the structure of the virus.) This can occur in childbirth as well as through blood transfusions or organ transplants prior to 1985. (Testing of the blood supply began in 1985, and the chance of this has greatly decreased.) Using injection equipment that an infected person used is another direct way to transmit HIV.

Sexual contact is also an effective transmission route for HIV because the tissues of the anus, rectum, and vagina are mucosal surfaces that can contain infected human body fluids and because these surfaces can be easily injured, allowing the virus to enter the body. A person is about five times more likely to contract HIV through anal intercourse than through vaginal intercourse because the tissues of the anal region are more prone to breaks and bleeding during sexual activity (Royce et al., 1997).

A woman is eight times more likely to contract HIV through vaginal intercourse if the man is infected than in the reverse situation (Center for AIDS Prevention Studies, 1998). HIV can be passed from a woman to a man during intercourse, but this is less likely because the skin of the
penis is not as easily damaged. Female-to-female transmission of HIV apparently is rare but should be considered a possible means of transmission because of the potential exposure of mucous membranes to vaginal secretions and menstrual blood (CDC, 1997a).

Oral intercourse also is a potential risk but is less likely to transmit the disease than anal or vaginal intercourse. Saliva seems to have some effect in helping prevent transmission of HIV, and the oral tissues are less likely to be injured in sexual activity than those of the vagina or anus. However, if a person has infections or injuries in the mouth or gums, then the risk of contracting HIV through oral sex increases.

Role of circumcision in male infectivity

A possible link between male circumcision and HIV infectivity was first observed during studies conducted in Kenya in the late 1980s (Cameron et al., 1998; Greenblatt et al., 1988; Simonsen et al., 1988). Since then, numerous studies have been done on the possible relationship between male circumcision and HIV infectivity. Data have not revealed a direct causal link between circumcision and HIV transmission, and scientific opinion has been divided on this topic. While some studies indicate that circumcision can play a protective role in preventing HIV infection (Kelly et al., 1999; Moses et al., 1998; Urassa et al., 1997), the bulk of recent scientific research has concluded that the reverse is true and that circumcision can actually increase the rate of HIV transmission (Van Howe, 1999). Clearly, further research and analysis of circumcision as a prophylactic against HIV transmission is needed.

Risks of transmission

Several factors can increase the risk of HIV transmission. One factor is the presence of another STD (e.g., genital ulcer disease) in either partner, which increases the risk of becoming infected with HIV through sexual contact. This is because the same risk behaviors that resulted in the person contracting an STD increase that person’s chance of contracting HIV. STDs also can cause genital lesions that serve as ports of entry for HIV, they can increase the number of HIV target cells (CD4+ T cells), and they can cause the person to shed greater concentrations of HIV (CDC, 1998a). For this reason, all sexually active clients, especially women, should be checked regularly for STDs such as gonorrhea and chlamydia. Many STDs that cause symptoms in men are asymptomatic in women. When genital ulcers are treated and heal, the risk of HIV transmission is reduced.

Another factor that increases risk is a high level of HIV circulating in the bloodstream. This occurs soon after the initial infection and returns late in the disease. New drug therapy can keep this level (called viral load) low or undetectable, but this does not mean that other individuals cannot be infected. The virus still exists--it is simply not detectable by the currently available tests. Because the correlation between plasma and genital fluid viral load varies, transmission may still occur despite an undetectable serum viral load (Liuzzi et al., 1996).

Once HIV passes to an uninfected person who is not taking anti-HIV drugs, the virus reproduces very rapidly. It is known that drug-resistant viruses can be transmitted from one person to another. The treatment implications for a person infected with a drug-resistant virus are not yet
known, but treatment will likely be difficult.

There are many misconceptions regarding HIV transmission. For example, HIV is not passed from one person to another in normal daily contact that does not involve either exposure to blood or sexual contact. It is not carried by mosquitoes and cannot be caught from toilet seats or from eating food prepared by someone with AIDS. No one has ever contracted AIDS by kissing someone with AIDS, or even by sharing a toothbrush (although sharing a toothbrush still is not advised). Other misconceptions people may have include the following:

- "It can't happen to me."--HIV can infect anyone who has sex with, or shares injection equipment with, someone who is infected.
- "I would know if my sex partner (injection partner) were infected."--Most people infected with HIV do not look or feel sick and do not even know they are infected.
- "As long as I get treated for any sexual infections I pick up, I'll be safe."--No current form of treatment can cure or prevent HIV, and although treating other infections reduces risk, there is still a high chance of getting HIV through unprotected sex or sharing injection equipment.
- "If I'm only with one sexual partner, and don't share injection equipment, I don't need to worry about HIV."--This is true only if the partner is uninfected and has no ongoing risk of infection. If the partner is or becomes infected, then anyone who has sex with him or shares his injection equipment is at high risk for HIV, and the only way to detect infection is to be tested.
- "If I douche or wash after sex, I won't get HIV."--Douching and washing will not prevent HIV.
- "If I don't share my own syringe, I won't get HIV."--HIV can also be spread through shared cookers, filters, and the prepared drug.

Life Cycle of HIV

It is possible to prevent transmission even after exposure to HIV. In San Francisco, postexposure prophylaxis is being offered to people who believe they have high risk for HIV transmission because of exposure with a known or suspected HIV-infected individual. Treatment is started within 72 hours of exposure and includes combination therapy, which may include a protease inhibitor, for a period of 1 month and followup for 12 months.

Once an HIV particle enters a person's body, it binds to the surface of a target cell (CD4+ T cell). The virus enters through the cell's outer envelope by shedding its own viral envelope, allowing the HIV particle to release an HIV ribonucleic acid (RNA) chain into the cell, which is then converted into deoxyribonucleic acid (DNA). The HIV DNA enters the cell's nucleus and is copied onto the cell's chromosomes. This causes the cell to begin reproducing more HIV, and eventually the cell releases more HIV particles. These new particles then attach to other target cells, which become infected. Figure 1-2 illustrates how HIV enters a CD4+ T cell and reproduces.

Measuring HIV in the blood
Physicians can measure the presence of HIV in a person by means of (1) the CD4+ T cell count and (2) the viral load count. The CD4+ T cell count measures the number of CD4+ T cells (i.e., white blood cells) in a milliliter of blood. These are the cells that HIV is most likely to infect, and the number of these cells reflects the overall health of a person's immune system.

CD4+ T cells act as signals to inform the body's immune system that an infection exists and needs to be fought. Because HIV hides inside the very cells responsible for signaling its presence, it can survive and reproduce without the infected person knowing of its existence for many years. Even though the body can produce sufficient CD4+ T cells to replace the billions that are destroyed by untreated HIV each day, eventually HIV kills so many CD4+ T cells that the damaged immune system cannot control other infections that may make the person sick. This is the late stage of HIV, when AIDS is often diagnosed based on the presence of specific illnesses (i.e., opportunistic infections).

The viral load represents the level of HIV RNA (genetic material) circulating in the bloodstream. This level becomes very high soon after a person is initially infected with HIV, then it drops. Viral load tests measure the number of copies of the virus in a milliliter of plasma; currently available tests can measure down to 50 copies per milliliter, and even more sensitive tests can measure down to 5 copies per milliliter.

To explain the relationship between CD4+ T cell count and viral load count and how together they are used to gauge a person's stage in disease progression, a "moving train" analogy can be used. The CD4+ T cell count is used to measure the person's distance to the point of high risk of contracting opportunistic infections, or death. The viral load count is used to measure the rate at which CD4+ T cells are being destroyed. Therefore, the CD4+ T cell count is the train's position on the track, and the viral load is the train's speed toward the outcome (i.e., AIDS and then death).

After a person is infected with HIV, the body takes about 6 to 12 weeks and sometimes as long as 6 months to build up proteins to fight the virus. These proteins are called HIV antibodies (disease-fighting proteins) and are detected by an HIV test called the ELISA (enzyme-linked immunosorbent assay). The ELISA is very sensitive—it almost always detects HIV if it is there. Rarely, ELISA tests will give false-positive readings (a positive test in someone uninfected). For this reason, a positive ELISA test must always be confirmed with a second, more specific test called the Western blot. According to the CDC, the accuracy of the ELISA and the Western blot together is greater than 99 percent. Rapid HIV tests and home sample collection tests also are options for clients; see Chapter 2 for a more detailed discussion of these types of tests.

The 6 to 12 weeks between the time of infection and the time when an ELISA test for HIV becomes positive are called the "window period." During this period, the individual is extremely infectious to any sexual or needle-sharing partner but does not test positive unless a more expensive viral load test is performed.

The level of virus is determined by using a viral load test; three types of viral load tests are HIV-RNA polymerase chain reaction (PCR), HIV branched DNA (bDNA), and HIV-RNA nucleic
acid sequence-based amplification (NASBA). Each of these tests measures the amount of replicating or reproducing virus in the bloodstream; thus a lower value signifies less risk of rapid progression. The best viral load test result is "none detected," although this does not mean the virus is gone, only that it is not actively reproducing at a measurable level.

**Disease Progression**

Once a person is infected with HIV, she should understand the progression of the disease from initial infection, through the latency period, symptomatic infections, and finally AIDS. The course of untreated HIV is not known but may go on for 10 years or longer in many people. Several years into HIV infection, mild symptoms begin to develop, then later severe infections that define AIDS occur. Treatment appears to greatly extend the life and improve the quality of life of most patients, although estimating survival after an AIDS diagnosis is inexact.

**Initial infection**

Primary HIV infection can cause an acute retroviral syndrome that often is mistaken for influenza (the flu), mononucleosis, or a bad cold. This syndrome is reported by roughly half of those who contract HIV (Russell and Sepkowitz, 1998) and generally occurs between 2 and 6 weeks after infection. Symptoms may include fever, headache, sore throat, fatigue, body aches, weight loss, and swollen lymph nodes. Other symptoms are a rash, mouth or genital ulcers, diarrhea, nausea and vomiting, and thrush. The CD4+ T cell count can drop very low during the early weeks, although it usually returns to a normal level after the initial illness is over. The initial illness can last several days or even weeks.

The greatest spread of HIV occurs throughout the body early in the disease. Approximately 6 months after infection, the level of virions produced every day may reach a "set point." A higher set point usually means a more rapid progression of HIV disease. Early treatment may be recommended to reduce the set point, potentially leading to a better chance of controlling the infection.

Alcohol and drug counselors should discuss symptoms that suggest initial HIV infection with their clients and encourage clients to be tested for HIV if they experience such symptoms. This not only will encourage clients who are infected to enter treatment early but also will provide an opportunity for the counselor to help uninfected clients remain that way.

**Latency period**

After initial infection comes the latency period, or incubation period, during which untreated persons with HIV have few, if any, symptoms. This period lasts a median of about 10 years. The most common symptom during this period is lymphadenopathy, or swollen lymph nodes. The lymph nodes found around the neck and under the arms contain cells that fight infections. Swollen lymph nodes in the groin area may be normal and not indicative of HIV. When any infection is present, lymph nodes often swell, sometimes painfully. With HIV, they swell and tend to stay swollen but usually are not painful.

**Early symptomatic infection**
After the first year of infection, the CD4+ T cell count drops at a rate of about 30 to 90 cells per year. When the CD4+ T cell count falls below 500, mild HIV symptoms may occur. Many people, however, will have no symptoms at all until the CD4+ T cell count has dropped very low (200 or less). Bacteria, viruses, and fungi that normally live on and in the human body begin to cause diseases that are also known as opportunistic infections.

Early symptoms of infection may include chronic diarrhea, herpes zoster, recurrent vaginal candidiasis, thrush, oral hairy leukoplakia (a virus that causes white patches in the mouth), abnormal Pap tests, thrombocytopenia, or numbness or tingling in the toes or fingers. Most of these infections occur with a CD4+ T cell count between 200 and 500. Symptoms of these infections usually signal a problem with the immune system but are not severe enough to be classified as AIDS. Please refer to Appendix D for a complete checklist of symptoms.

**AIDS**

In the 1980s, AIDS was defined to include a depressed immune system and at least one illness tied to HIV infection. AIDS-defining conditions are diseases not normally manifest in someone with a healthy immune system. These should prompt a confirmatory HIV test. The additional 1993 AIDS-defining conditions led to the diagnosis of more AIDS cases in women and injection drug users. Since 1993, the list of AIDS-defining conditions has included pulmonary tuberculosis (TB), recurrent bacterial pneumonia, and invasive cervical cancer. HIV-infected persons with a CD4+ T cell count of 200 or less are classified as persons with AIDS (CDC, 1992).

TB and invasive cervical cancer are two AIDS-defining conditions that warrant special mention. Pulmonary TB is the one AIDS-related infection that is contagious to those without HIV. It generally causes a chronic dry cough (sometimes with blood), fatigue, and weight loss. Pulmonary TB requires ongoing treatment for at least 6 months, and close associates of the infected person must be tested for TB. If TB is only partially treated (i.e., the TB patient does not take all of the medications), resistant TB will develop, which can then be passed to others. Although TB, coupled with a positive HIV test, is an AIDS-defining diagnosis, it also can occur while the CD4+ T cell count is still high. If TB occurs late in the disease after the CD4+ T cell count has dropped, it may not be found in the lungs, and symptoms may include only weight loss and fever, without a cough. It should be noted, however, that the Mantoux PPD test (a test routinely administered to screen for TB by determining reaction to intradermal injection of purified protein derivative) may not be positive if the patient is anergic (i.e., if he has sufficient immune system damage to cause inability to respond to the PPD).

Cervical cancer may progress rapidly in women with HIV but usually is asymptomatic until it is too late for successful treatment. Women who are HIV positive should have Pap tests at least once every 6 months and more often if any abnormality is found.

**AIDS symptoms**

Most AIDS-defining diseases are severe enough to require medical care, sometimes hospitalization. Some of these diseases, however, can be treated earlier on an outpatient basis if
symptoms are reported when they are mild. (Please refer to Appendix C for a complete list of AIDS-defining conditions.)

Cough is a symptom common to several AIDS-related infections, the most frequent of which is *Pneumocystis carinii* pneumonia (PCP—not to be confused with the drug by that name, phencyclidine). PCP is characterized by a dry cough, fever, night sweats, and increasing shortness of breath. Recurrent bacterial pneumonia (i.e., two or more infections within a year) also is an AIDS-defining condition. It often causes a fever and a cough that brings up phlegm. Coughing is also a symptom of TB. As a general guideline, if a cough does not resolve after several weeks, it should be checked by a medical practitioner.

Several skin problems can occur in HIV/AIDS. Kaposi's sarcoma (KS), a rare malignancy outside of HIV disease, may be the best-known skin condition in HIV infection. KS is a cancer of the blood vessels that causes pink, purple, or brown splotches, which appear usually as firm areas on or under the skin. KS also grows in other places, such as the lungs and mouth. KS is highly prevalent among men with AIDS, of whom 20 to 30 percent may develop the condition in contrast to 1 to 3 percent of women with AIDS (Kedes et al., 1997). However, since the introduction of combination anti-HIV therapy, KS is seen less frequently.

Diarrhea is a very common symptom of AIDS. Many AIDS-defining conditions cause diarrhea, including parasitic, viral, and bacterial infections. HIV itself can cause diarrhea if it infects the intestinal tract. Diarrhea also is a common side effect of HIV/AIDS medications. Weight loss can be caused by inadequate nutrition, untreated neoplasms and opportunistic infections (which often are associated with diarrhea), and deranged metabolism (Dieterich, 1997).

Changes in vision, particularly spots or flashes (known as "floaters"), may indicate an infection inside the eye. A virus called cytomegalovirus (CMV) is the most common cause of blindness in people with HIV/AIDS. CMV progresses very rapidly if not treated and is among the most feared of AIDS-related infections. Fortunately, it almost never occurs until the immune system is almost completely destroyed, so it is not usually the first symptom. Counselors can screen for early signs of CMV using the Amsler Grid (see Appendix D). The client also can be taught to screen himself using this screening tool.

A severe headache, seizure, or changes in cognitive function may herald the onset of a number of infections or cancers inside the brain. The two most common brain infections in HIV/AIDS are cryptococcal meningitis, a fungus that usually causes a severe headache, and toxoplasmosis, which can present with focal neurologic deficits or seizure. Seizures also can be caused by the cancer of the central nervous system called lymphoma. Progressive multifocal leukoencephalopathy (PML), a brain disease that causes thinking, speech, and balance problems and dementia also can occur as a result of HIV infection.

End-stage disease

A person with HIV/AIDS can live an active and productive life, even with a CD4+ T cell count of zero, if infections and cancers are controlled or prevented. The newer antiviral medicines can even help the body restore much of its lost immune function. In the past few years, a
phenomenon called the Lazarus syndrome has developed among patients with AIDS, wherein, because of optimal drug therapy, someone who had seemed very near death improves and returns to fairly normal function. Untreated, the disease eventually overwhelms the immune system, allowing one debilitating infection after another. Sometimes the possible combinations of medication are no longer effective, the side effects are intolerable, or no further therapy is available.

Hospice care is an appropriate choice for those who have run out of therapeutic options. In hospice care, the individual is treated for pain and other discomforts and allowed to die of the disease. Pain therapy at this stage invariably requires narcotics. It is crucial that the client and other treatment professionals understand that using opiates for pain is entirely different from using them to feed an addiction. The client will develop a need for high doses and will have withdrawal symptoms if the drug is stopped, but will not "get high." If drugs must be stopped (which is uncommon), they can be tapered under medical supervision. See Chapter 2 for a more in-depth discussion of pain management.

Hospice care allows the person with end-stage HIV/AIDS a peaceful death and a chance to address those relationships or experiences that are important. Hospice goals involve maintaining dignity and allowing the client’s significant others to dictate how they will cope with this final stage.

Changes in the Epidemiology of HIV/AIDS Since 1995

With the advent of new and effective treatments, the epidemiology of HIV/AIDS is changing. The study of HIV/AIDS epidemiology helps to identify the trends of the disease. Surveillance of AIDS cases since 1996 shows substantial declines in AIDS-related deaths and increases in the number of persons living with AIDS, although the decline is slowing (CDC, 1999b). As people live longer with HIV/AIDS, the ability to use AIDS surveillance data alone to represent trends has diminished. It is difficult but important to track the distribution of prevalence (i.e., existing) and incidence (i.e., new) of both HIV and AIDS cases to detect changes in geographic, demographic, and risk/exposure trends (Ward and Duchin, 1997-1998).

With the mid-year 1998 edition, the CDC started to include information from both HIV infections and AIDS cases in the HIV/AIDS Surveillance Report (CDC, 1998c). It should be noted that the number of HIV cases in the report is a conservative estimate of the number of people living with HIV because not all people with HIV/AIDS have been tested (and those who have been tested anonymously are not reported to State health departments' confidential, name-based HIV registries). At the end of June 1999, 30 States and the U.S. Virgin Islands were reporting HIV cases.

This section presents an overview of the trends in the HIV/AIDS pandemic and discusses how the pandemic intertwines with substance abuse. The information is organized to provide a general look at the pandemic in the United States and its Territories, a discussion of the trends and the populations which are most at risk for contracting the infection, and a regional look at the pandemic (the regions are defined by the CDC). Finally, there is a discussion of special populations and how they are affected by the HIV/AIDS pandemic. For more detail about

To see the distribution of HIV/AIDS in the United States, see Figures 1-3 through 1-6. Figure 1-3 shows the AIDS rates for male adults and adolescents reported from July 1998 through June 1999. Figure 1-4 shows the number of adult and adolescent male AIDS and HIV cases reported from July 1998 through June 1999. Figure 1-5 illustrates the AIDS rate for female adults and adolescents reported from July 1998 through June 1999, and Figure 1-6 shows the number of female adult and adolescent AIDS and HIV cases reported from July 1998 through June 1999.

**Current Trends in the HIV/AIDS Pandemic**

Current trends in HIV/AIDS disproportionally affect racial minority populations, especially women, youth, and children within those populations. HIV prevalence is higher among African Americans than in other ethnic groups; from July 1998 through June 1999, African Americans accounted for 46 percent of adult AIDS cases, while representing 12 percent of the total U.S. population. Hispanics accounted for 20 percent of adult AIDS cases from July 1998 through June 1999, while making up only 11 percent of the total U.S. population (CDC 1999b; U.S. Bureau of the Census, 1998). Together, African Americans and Hispanics represent the majority of AIDS cases thus far in the pandemic (CDC, 1999b, 1999c). In addition, of the HIV cases reported from the 30 States and one Territory from July 1998 through June 1999, 54 percent were among adult and adolescent African Americans, and 10 percent were among adult and adolescent Hispanics. Substance abuse is a primary mechanism by which these vulnerable groups become HIV-infected populations.

It is important to be aware that, although it is customary to categorize cases based on broad ethnic labels, this procedure glosses over fundamental ethnic and cultural differences among people of color and fails to address the underlying economic and social infrastructure that fuels the spread of substance abuse and HIV (National Commission on AIDS, 1992). Categorizing all persons with African racial heritage as "black" mixes together people of distinct ethnic and cultural heritage (e.g., ethnic descendents of African slaves, Caribbean immigrants) as well as individuals from different socioeconomic groups. Similarly, "Hispanic" refers to a multiethnic and multicultural blend of people from more than 30 geographic regions. Social, political, and economic forces have led to the "ghettoization" of African Americans and Hispanics in the inner cities where there are high rates of drug trafficking, unemployment, poverty, racism, and a lack of access to health care, all of which contribute to high rates of addiction and HIV/AIDS (National Commission on AIDS, 1992). It is within urban, poor, African American and Hispanic communities that HIV/AIDS is most prevalent.

These oppressive socioeconomic factors also have led to high rates of incarceration, sex work, and homelessness for members of African American and Hispanic communities. Drug offenses account for the highest number of Federal crimes for which people are incarcerated (Mumola, 1999). For example, a survey of new commitments to California State prisons found that more than 75 percent of the offenders had histories of drug use (California Department of Corrections,
Not surprisingly, these individuals also have high rates of HIV infection (Stryker, 1993). Sex workers, many of whom are poor, homeless, and substance dependent, are likely to be more concerned with immediate needs such as housing, food, or substance abuse than HIV or substance abuse prevention and intervention (Kail et al., 1995). This is also true for the homeless or marginally housed who often are dealing with both substance abuse and mental health or mental retardation problems (St. Lawrence and Brasfield, 1995).

However, the highest HIV and AIDS rates among at-risk populations are still found among MSMs (CDC, 1999b), who from July 1998 through June 1999 represented 38 percent of AIDS cases and 30 percent of HIV cases. Minority MSMs especially are at high risk for contracting the infection. See the section “HIV/AIDS Epidemiology Among Groups” later in this chapter for further discussion of HIV/AIDS and MSMs.

HIV/AIDS is epidemic among the heterosexual population as well and is fueled by sexual contact with HIV-infected, injection drug-using, or bisexual partners. Heterosexuals located in communities with high prevalence of HIV/AIDS and addiction are at greatest risk for contracting HIV/AIDS from heterosexual contact. This type of heterosexual contact, defined generally as sexual contact with an "at-risk" person (e.g., injection drug users, bisexual man) or an HIV-infected person whose risk was not specified, from July 1998 through June 1999 accounted for about 15 percent of all adult and adolescent AIDS cases and about 17 percent of reported adult and adolescent HIV infection cases (CDC, 1999b). Of these, 61 percent of AIDS cases were women and 39 percent were men; of HIV infection cases, 68 percent were women and 32 percent were men.

From July 1998 through June 1999, there were 4,296 new AIDS cases and 2,321 new HIV cases among women who reported heterosexual contact (CDC, 1999b). Of these, 28 percent of AIDS cases and 21 percent of HIV cases were among women who reported sexual contact with injection drug users, 5 percent of AIDS cases and 6 percent of HIV cases who reported sexual contact with bisexual men, and 66 percent of AIDS cases and 72 percent of HIV cases who reported sexual contact with an HIV-infected person, without reporting the origin of the partner's infection. Of the 2,754 AIDS cases and 1,070 HIV cases for men who reported heterosexual contact, the majority reported sexual contact with an HIV-infected person without reporting the origin of the partner's infection (77 percent of AIDS cases and 80 percent of HIV cases). These data are supported by earlier research that found that HIV infection among heterosexual clients in alcohol abuse treatment, who were primarily male, was largely caused by unsafe sexual behaviors (Avins et al., 1994; Woods et al., 1996).

Figures 1-7 and 1-8 illustrate the trend of male and female AIDS cases contracted through heterosexual exposure from 1993 to 1998 by ethnicity. These figures depict only self-identified heterosexual men and women.

Regional HIV/AIDS Epidemiology

Early in the U.S. AIDS pandemic, the Northeast region of the United States had the most AIDS cases, followed by the South, Midwest, and the West (Figure 1-9 contains a breakdown of the States that make up these four regions plus the U.S. Territories, as defined by the CDC). In all
regions, AIDS incidence increased through 1994, with the most dramatic increases occurring in the South. Between 1997 and 1998, AIDS incidence dropped for all regions, but in 1998 the South still had the highest rate (43 percent), followed by the Northeast (28 percent), the West (17 percent), the Midwest (8 percent), and the U.S. Territories (3 percent) (CDC, 1999b). Figure 1-10 demonstrates the change in AIDS incidence of the regions for 1996, 1997, and 1998.

The HIV/AIDS pandemic is evolving differently in different regions of the United States, just as drug use varies from region to region. Therefore, alcohol and drug counselors should become familiar with HIV/AIDS prevalence, incidence, and trends in their local areas, their States, and their regions. Appendix G contains a list of State and Territory departments of health (including addresses, phone numbers, and Web sites where readers can obtain information about their State). When available, State AIDS hotlines also are listed.

The 10 States and Territories reporting the most AIDS cases, in descending order, are New York, California, Florida, Texas, New Jersey, Puerto Rico, Illinois, Pennsylvania, Georgia, and Maryland. The 10 metropolitan areas reporting the highest number of AIDS cases, in descending order, are New York City, Los Angeles, San Francisco, Miami, the District of Columbia, Chicago, Houston, Philadelphia, Newark, and Atlanta (CDC, 1999b). Not surprisingly, these major metropolitan areas also are high-intensity drug-trafficking areas as defined by the Office of National Drug Control Policy (ONDCP, 1998).

HIV Epidemiology Among Groups

Homosexuals

The primary route of HIV transmission for MSMs is through sexual contact, which may occur while the participants are engaged in substance abuse, including IDU. Within this group, the focus of the pandemic among MSMs has shifted from older, white, urban men to poorer African American and Hispanic men, men with substance abuse problems (including IDU), and young men. Repeated studies have found that MSMs who abuse alcohol, speed, MDMA (3,4-methylenedioxymethamphetamine), cocaine, crack cocaine, inhalants, and other noninjection street drugs are more likely than those who do not use substances to engage in unprotected sex and become infected with HIV (Paul et al., 1991b, 1993, 1994). One hypothesis about the reason for higher rates of HIV/AIDS among MSMs is that substance abuse may increase sexual risktaking. This is because substance abusers experience decreased inhibition, new learned behaviors (such as using substances and then having unprotected anal intercourse), low self-esteem, altered perception of risk, lack of assertiveness to negotiate safe practices, and perceived powerlessness (Paul et al., 1993).

As of June 1999, more than half of all cumulative male adult and adolescent AIDS cases were among MSMs who reported sexual risk only (57 percent) or sexual risk and IDU (8 percent). Of cumulative HIV cases among adult and adolescent males, 45 percent reported sexual risk only and 6 percent reported sexual risk and IDU (CDC, 1999b). Even though the cumulative total of AIDS cases among MSMs is still highest in white men (62 percent white, 23 percent African American, 14 percent Hispanic), new AIDS cases among MSMs indicate that the disparity between cases among whites and among minorities is narrowing. From July 1998 through June
1999, 53 percent of AIDS cases were among white men, 29 percent were among African American men, and 16 percent were among Hispanic men. Figure 1-11 illustrates the trend of MSM AIDS cases by ethnicity from 1993 to 1998.

As with injection drug users, minority MSMSMs are disproportionately affected by HIV disease. African American and Hispanic MSMSMs, compared with their white counterparts, are more likely to inject drugs, to be substance abusers, to be poor, to be paid for sex, and to engage in higher rates of unprotected anal intercourse (National Commission on AIDS, 1992; Peterson et al., 1992). Sociocultural factors, combined with some community values (e.g., machismo, family loyalty, sexual silence) and lack of access to health care and substance abuse treatment, strongly compete with safe sex and drug practices among gay and bisexual men of color (Diaz and Klevens, 1997).

Sex networks and sexual mixing patterns (Renton et al., 1995) are hypothesized to explain the higher risk of HIV infection related to substance abuse among MSMSMs. MSM substance abusers may form tight groups characterized by higher HIV seroprevalence rates, higher sexual mixing, greater IDU, and more trading of sex for money, food, and drugs. These factors are another way to account for higher HIV risk-taking sexual behaviors among MSM substance abusers.

Incarcerated persons

A recent study reported that the confirmed rate of AIDS cases among incarcerated people in State and Federal prisons is more than six times higher than in the general population. About 2.3 percent of all persons incarcerated in the United States in 1995 were HIV positive, and about 0.51 percent had confirmed AIDS (MacDougall, 1998; Maruschak, 1997). According to the Bureau of Justice Statistics in the U.S. Department of Justice, in 1997, 57 percent of State prisoners and 45 percent of Federal prisoners said they had used drugs in the month before committing their offense. In addition, 83 percent of State prisoners and 73 percent of Federal prisoners said they had used drugs at some time in the past. Even with these high rates, which increased between 1991 and 1997, substance abuse treatment services declined during the same time period (Mumola, 1999).

In 1991, only 1 percent of Federal prison inmates with substance abuse disorders received appropriate treatment. For those who completed treatment there were no aftercare services in place to help them remain abstinent after they got out of prison (U.S. General Accounting Office, 1998).

Most incarcerated people who have HIV are infected before they enter prison. One study of 46 prisons found an HIV infection rate of 1.7 percent among people entering prison (Withum, 1993). In some correctional facilities, HIV infection rates are as high as 20 percent among women and 15 percent among men. For MSMSMs, HIV infection rates ranged from 9 to 34 percent; among injection drug users the infection rate ranged from 6 to 43 percent.

HIV/AIDS and substance abuse interventions implemented in prisons have a great potential to impact the HIV/AIDS pandemic (MacDougall, 1998). Like the HIV-infected population, the incarcerated population has an overrepresentation of minority groups and is characterized by
high poverty, overcrowding, IDU, high-risk sexual activities, and poor access to health care. Incarceration presents an opportunity to screen, counsel, and educate inmates about HIV/AIDS, and to provide substance abuse treatment as well. For many incarcerated persons, this may be their first contact with medical interventions as well as with substance abuse treatment.

When prison inmates return to society, their health status will have an effect on the community to which they return. A study of Hispanic inmates in California found that 51 percent reported having sex within the first 12 hours after release and that they preferred not to use condoms (Morales et al., 1995). In addition, 11 percent reported IDU in the first day after release.

Sex workers

The sex workers who are most vulnerable to contracting and transmitting HIV are street workers, who often are poor or homeless, may have a history of childhood abuse, and are likely to be alcohol or drug dependent. A CDC study of female sex workers in six U.S. cities found an HIV seroprevalence of 12 percent, ranging from 0 to 50 percent depending on the city and the level of IDU (CDC, 1987a). A study of male sex workers in Atlanta found an HIV seroprevalence of 29 percent, with the highest rates among those who had receptive anal sex with nonpaying partners (Elifson et al., 1993).

IDU was the main risk factor for HIV infection for female sex workers in six U.S. cities (CDC, 1987a). Female injection drug users who trade sex for money or drugs are more likely to share needles than female injection drug users who do not engage in sex trading (Kail et al., 1995). The circumstances in which sex workers live also increase their chances of contracting HIV. For example, they may agree to unprotected sex if a client offers more money, if they are desperate for money to buy drugs, or if business has been slow. Violent clients may force unsafe sex, and in many cities police confiscate condoms when they arrest or stop sex workers. HIV prevention outreach to sex workers is difficult because prostitution is illegal. Immediate attention to concerns about food, housing, and drug addiction often take precedence over HIV prevention.

Homeless or marginally housed

Homelessness often occurs in conjunction with substance abuse, chronic mental illness, and unsafe sexual behavior. All of these factors increase homeless people’s risk for contracting HIV. A survey of 16 U.S. cities found that 3 percent of homeless people were HIV positive, compared with less than 1 percent of the general adult population (Allen et al., 1994). In other studies, 19 percent of homeless mentally ill men in New York City were HIV positive (Susser et al., 1993), and an 8 percent HIV infection rate was found among homeless adults in San Francisco (Zolopa et al., 1994).

A survey of homeless adults in a storefront medical clinical found that 69 percent were at risk for HIV because of the following factors: (1) unprotected sex with multiple partners, (2) IDU, (3) sex with an injection drug-using partner, or (4) exchanging unprotected sex for money or drugs. Almost half reported at least two of these risk factors, and one fourth reported three or more risk factors (St. Lawrence and Brasfield, 1995). Substance abuse can exacerbate HIV risks because abusers are more likely to forget to use condoms, to share needles, and to exchange sex for
drugs. A survey of homeless adults in St. Louis found that 40 percent of men and 23 percent of women reported drug use, and 62 percent of men and 17 percent of women reported alcohol use (North and Smith, 1993).

Adolescents

Because the average period of time from HIV infection to AIDS is about 10 years, most young adults with AIDS were likely infected as adolescents (National Institute of Allergy and Infectious Diseases [NIAID], 1999). Through June 1999 in the United States, 3,564 cases of AIDS in people aged 13 through 19 were reported (CDC, 1999b). In the 13- to 19-year-old age group, 60 percent were male and 40 percent were female. When broken down by ethnic group, 30 percent were white, 49 percent were African American, 20 percent were Hispanic, and 1 percent were Asian/Pacific Islander or American Indian/Alaskan Native.

Most adolescents are exposed to HIV through unprotected sex or IDU. Through June 1999, HIV surveillance data show that there were 4,470 cases reported in the 13- to 19-year-old age group. Of those, 45 percent were male, and 55 percent were female. When broken down by ethnic group, 27 percent were white, 66 percent were African American, 5 percent were Hispanic, and less than 1 percent each were Asian/Pacific Islander or American Indian/Alaskan Native (CDC, 1999b). Half of the infected male adolescents reported exposure through sex with men.

Almost half (42 percent) of female adolescents were exposed to HIV through heterosexual contact. Another significant trend is the number of STDs reported among adolescents: About two thirds of the 12 million cases of STDs reported in the United States each year are among individuals under the age of 25, and one quarter are among teens. This is significant because the presence of an STD can increase the risk of HIV transmission threefold to ninefold, depending on the type of STD (NIAID, 1999).

Adolescents tend to believe they are "invincible" and therefore engage in risky behaviors. Because of this belief they also may delay HIV testing, and, if they do test and are positive, they may delay or refuse treatment. Alcohol and drug counselors who work with adolescents should encourage them to be tested for HIV if they are at risk. Adolescents can be helped by having information about HIV/AIDS explained to them clearly, by drawing out information about behaviors that may have put them at risk for HIV, and by emphasizing the success of newly available treatments.
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Human Immunodeficiency Virus Infection Prevention: Strategies for Clinicians

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The complexity of the epidemic of human immunodeficiency virus (HIV) infection has made the creation of effective prevention programs an evolving and challenging task. Prevention of new HIV infections is an issue of increasing importance as the prevalence of HIV infection continues to increase. The integration of prevention and clinical care is recognized as a key element of future prevention activities. Prevention of HIV infection should extend beyond the traditional public health model to the clinical care site. The clinical care setting offers a unique opportunity to bring people with HIV disease into care and to establish relationships, thus creating a foundation for prevention-related activities. However, the expertise and energy that clinicians currently dedicate to diagnosis and treatment far exceeds that directed toward prevention. We review details about and the efficacies of specific prevention efforts of relevance in clinical practice, and we conclude with practical recommendations regarding the most simple and efficient ways of integrating both behavioral counseling and medical interventions as prevention tools into clinical practice.

Reports from the 14th International AIDS Conference (Barcelona, Spain) project that, in the absence of an expanded prevention effort, there will be 45 million new HIV infections by 2010. It is estimated that 29 million of these infections could be prevented with the expansion of existing prevention strategies. The success of prevention programs in both developing (e.g., Senegal, Thailand, and Uganda) and developed (e.g., United States) countries indicates that prevention does work. Although the bulk of our experience has involved the HIV-seronegative population, more recent strategies have shifted focus to the HIV-seropositive population [1, 2]. Of particular concern (and our focus here) is the population of HIV-seropositive patients in the clinical care setting. It is these patients who may be receiving antiretroviral therapy (ART) and exhibiting high-risk behaviors and thus have the potential to transmit both drug-susceptible and drug-resistant virus. This review will summarize the experience, to date, on HIV prevention in the clinical care setting and on how we can use this experience to best incorporate prevention into our practice (table 1).

RISK BEHAVIORS

Although millions of people are at behavioral risk for HIV disease, transmission can occur only through people who are currently infected with HIV. A substantial minority of people living with HIV infection continue to engage in both high-risk sexual and high-risk drug use behaviors. Although the numbers vary greatly among different populations, continued risky behaviors have been documented among all subgroups of HIV-infected individuals: injection drug users (IDUs), heterosexual men and women, and men who have sex with men (MSM) [3–5]. Although much of the previous research on risk behavior has focused on sexual risk behaviors among MSM [6, 7], risky behavior affects all HIV-infected patient groups, including IDUs and heterosexual men and women [8].

Previous studies involving MSM have found that risk behaviors may increase if patients receiving ART have subjectively improved feelings of well-being or believe that an undetectable virus load indicates that it is safe to engage in high-risk behavior [9–13]. Trends in risk behavior and their relationship to receipt of ART may differ in HIV-positive IDUs or heterosexuals, who may have different cultural backgrounds and varied levels of...
TRANSMISSION OF DRUG-RESISTANT HIV

Transmission of drug-resistant HIV has been well documented among all patient groups, including MSM, IDUs, heterosexuals, infants, and health care workers [14–18]. A recent report by Little et al. [19] documented a marked increase in the prevalence of primary genotypic (23%) and phenotypic (12.4%) drug resistance among 377 patients who had seroconversion in the 12 months before the study, when compared with the prevalence in 1995. Data regarding the prevalence of drug resistance among patients with chronic HIV infection in the era of HAART was recently reported [20, 21]. The largest study indicated a 78% prevalence of drug resistance among patients who were receiving HAART. This is of particular concern with regard to the prevention of HIV infection, because it is this group of patients that are capable of transmitting drug-resistant virus to uninfected persons [22].

PREVENTION IN THE CLINICAL CARE SETTING

Behavioral Counseling

**Past experience.** There is substantial evidence that behavioral counseling works as a prevention strategy [23–25]. The National Institute of Mental Health (NIMH) Multisite HIV Prevention Trial [26] assessed the efficacy of a motivational behavioral intervention to reduce sexual risk behaviors for HIV infection among high-risk, heterosexual, low-income patients at 37 clinics across the United States. Patients in the intervention group who underwent a 7-session HIV infection risk–reduction program over the course of 1 year subsequently reported fewer unprotected sexual acts, had higher levels of condom use, and were more likely to use condoms consistently during a 12-month follow-up period. Other studies that used a 5-session intervention delivered by community-based para-professionals or mental health counselors [23] and an 8-week course of sex-specific counseling sessions [24] have shown significant benefit in reducing risk behaviors for HIV transmission.

Although previous studies had shown the efficacy of counseling on the initiation of behavior changes, Kamb et al. [27] conducted the first large, randomized, controlled trial (Project Respect) to evaluate the effect of counseling in reducing sexually transmitted diseases (STDs) among HIV-negative heterosexual patients at an STD clinic. The results showed that subjects in the 2 counseling arms were significantly more likely than those in the education-only arm to use condoms 100% of the time at 3 months of follow-up and were 30% less likely to have an incident STD (on the basis of laboratory diagnosis) at 6 months of follow-up.

In summary, previous studies indicate that behavioral counseling can indeed reduce sexual risk behaviors. However, the majority of these studies have used risk reduction counseling by specialized and trained research staff and not the usual clinical providers. It is imperative to broaden and generalize these experiences by devising prevention instruments and techniques for clinicians to use in the usual clinical care setting.

**Barriers.** Although clinicians believe that health promotion and disease prevention are part of the job, they currently engage in very little risk reduction intervention activity [28]. Barriers to clinician-delivered interventions are varied and include lack of training about and knowledge of sex- and drug-related behaviors, lack of discussion skills and reluctance to discuss issues of sex and drug use, belief that their attempts will not be successful, absence of perception that their patients are at risk, lack of standardized tools to assess patient risk, and, finally, constraints of time and resources. Clinicians may find it uncomfortable to discuss such issues as sex and prevention of infection, or they may believe that their patients are uncomfortable discussing these issues. Others may be busy discussing issues of adherence, toxicities, laboratory monitoring, and health maintenance and feel that there may not be sufficient time to address the issue of prevention. Still others may unrealistically perceive the goal of prevention activities to be the elimination of all high-risk behaviors, with an expectation of complete abstinence or sobriety rather than the aim of attain-

### Table 1. Strategies for clinicians.

| Behavioral counseling interventions, with the following parameters |
|--------------------------|--------------------------|
| Supportive and nonpunitive counseling in a comfortable setting |
| Interactive counseling and motivational interviewing |
| Individualized counseling |
| Scripting of conversations |
| Goal-directed counseling with reasonable, attainable goals |
| Repeated sessions and incorporation into every clinical encounter |
| Medical interventions |
| Antiretroviral therapy |
| Reduction to and maintenance of undetectable virus load |
| Drug resistance monitoring |
| Promotion of adherence to therapy |
| Postexposure prophylaxis |
| Screening, diagnosis, and treatment of sexually transmitted diseases |
| Public health interventions |
| Promotion and distribution of condoms and clean needles |
| Use of HIV infection prevention posters, fact sheets, and brochures |
| Partner counseling and notification |
| Referral services |
| Substance abuse treatment |
| Psychiatric treatment |
able and stepwise risk reduction; thus, they believe that their efforts will be fruitless.

Research, however, indicates that patients view clinicians as a trusted source of prevention information [29], and data on interventions for other behavioral issues show that a clinician’s prevention messages can be effective. Success in changing patient behavior has been documented in many instances, including exercise promotion [30], smoking cessation [31], coronary risk reduction [32], breast self-examination [33], and adherence to STD treatment [34]. In addition, a wide variety of prevention programs have shown success in changing clinicians’ behavior with their patients. The techniques described below can be used to overcome the aforementioned barriers and ideally can lead to positive changes in both patient and clinician behaviors.

**Recommendations to clinicians.** HIV disease is a chronic illness requiring multiple clinical encounters and a close patient-provider relationship. Furthermore, the clinic may be the only place where HIV-infected patients will have contact with someone who can inform and educate them about prevention of HIV transmission. The skills of clinicians and the relationships that they develop over time with patients can be favorably used as a key to a prevention strategy. Our personal experience with the NIMH-funded Options Project [35, 36] is an example of how prevention can be integrated into the clinical care setting. The Options Project involves clinicians’ use of motivational interviewing techniques [37] at each clinical encounter to promote HIV risk reduction behavior change. The motivational interviewing strategies used include assessing the patients’ HIV transmission risk behaviors and asking the patients to rate, on 10-point scales, how important it would be for them to reduce their risk behavior (or to maintain their pattern of safer behavior) and how confident they are that they could reduce their risk behavior or maintain safer practices. The patient and clinician then together devise prevention strategies to improve these scores. This encounter is repeated at every clinic session, and it is always tailored to the patient’s ongoing transmission risk behaviors. Preliminary evidence suggests that the Options Project intervention may be effective in reducing risk behaviors for HIV transmission among HIV-infected patients receiving clinical care and that the intervention appears to have a significant impact on assisting individuals to maintain no risk [36]. We thus recommend behavioral counseling as a prevention tool in the clinical care setting, with the parameters that follow.

1. **Supportive and nonpunitive counseling in a comfortable setting.** Surveys indicate that patients do want to discuss such issues as sex and HIV prevention with their clinician and that they actually expect these discussions to take place. Creating a comfortable, nonjudgmental atmosphere in which the patient can discuss these issues is critical for the clinician and patient to gain insight and to learn the best strategies for HIV prevention for each particular patient.

2. **Interactive counseling or motivational interviewing.** The content and duration of counseling necessary to achieve meaningful change in risk behaviors is an evolving issue. Strategies focusing on behavioral change have evolved over time as various tactics, which vary from a didactic educational approach to the more recent interactive counseling approach, have been used. The technique of motivational interviewing can help the patient discuss behavior and be involved in the decision-making process. It encourages patients to describe their behaviors and develop their own solutions. It can open up the door to productive discussions and use the patients’ own strengths and views as tools to help them arrange safer behavior.

3. **Individualized counseling.** Given the variability of patients and settings, it is difficult to design a single effective intervention to be used in the clinical care setting. Recognizing the specific needs for each patient is critical in terms of formulating a plan for prevention. Although some patients may not be engaging in any high-risk behaviors, this may change over time, and it must be assessed at each visit. Each clinician must take into account what he or she knows about an individual patient, both from a behavioral and a biological standpoint. The technique of motivational interviewing is useful in that it can be applied and tailored to meet the needs of any particular patient.

4. **Scripting of conversations.** Initiating and incorporating discussions of sexual and drug use behavior is often difficult for clinicians. However, there are techniques that can be used to overcome this barrier. One used in the Options Project is the use of scripted phrases. Some clinicians find it helpful to have a scripted conversation to initiate discussion. This can help both by serving as a reminder to the clinician and by providing a way for the clinician to feel more comfortable introducing these issues. Furthermore, it can help provide the most sensitive way to deliver messages.

5. **Goal-directed counseling.** It is important that patients have a concrete sense of what they can do at the individual level to prevent the spread of HIV disease. Together with clinicians, patients can come up with goals that would specifically apply to their own risk of transmitting HIV. For example, a patient may agree to use condoms consistently or agree to always inform partners of his or her serostatus. In addition to the traditional therapeutic prescription that usually closes each clinical encounter, a tactic used in the Options Project is to hand each patient a “prevention prescription” with his or her individual goal for prevention of the spread of HIV infection.

6. **Repeated sessions.** Studies have shown that, although prevention messages may be part of initial encounters, they are much less frequently incorporated into subsequent visits. However, prevention messages need to be repeatedly delivered to
be effective. In addition, patients’ behaviors change over time as their disease courses and social situations vary, which further emphasizes the importance of tailoring prevention messages to a particular point in time. There is no urgency to prevention messages; more importantly, there is a need to repeatedly deliver these messages as a routine part of every clinical encounter.

For example, a patient may be asked, “Now that we’ve finished discussing your medications, I’d like to ask you some questions about your sex and drug use behaviors. What behaviors are you involved in now? Would you feel comfortable discussing them? Can you think of anything that you might like to change about these behaviors, and what interest might you have for changing them? How might you be able to reduce the riskiness of your sex and drug use behaviors?” Or the patient may be asked, “How important is reducing risk behavior to you (on a scale of 1 to 10), and how confident are you that you can do this (on a scale of 1 to 10)?” These are general questions that allow clinicians to draw patients out and encourage them to lead the discussion. They are the most knowledgeable about the details of their behavior, and, with help to think it through, they will often come up with their own solutions.

**Medical Interventions**

In addition to behavioral counseling, traditional medical interventions including administration of HAART, administration of postexposure prophylaxis (PEP), promotion of adherence to therapy, and diagnosis and treatment of STDs are essential parts of a clinician’s prevention armamentarium.

**ART and PEP.** ART has the potential to reduce virus load, infectiousness, and the likelihood of HIV transmission [38], thus theoretically functioning both as a treatment modality and as a preventive tool. The impact that ART can have on reducing the risk of HIV transmission has been demonstrated in the prevention of mother-to-child transmission of HIV [39, 40]. It follows from this experience that ART could also be of potential benefit in reducing the likelihood of HIV transmission by sexual and bloodborne transmission. Although US Centers for Disease Control and Prevention policy advocates the use of ART for PEP after occupational exposure to HIV [41], the primary evidence for the effectiveness of PEP in preventing sexual transmission of HIV derives from animal studies and extrapolation of data on occupational exposure [42], and the strategy has not yet been proven to be effective. ART and PEP are currently areas of great interest and potential benefit with regard to prevention of HIV transmission; however, both await further study before their utility as preventive measures can be established.

**Adherence to therapy.** Adherence is a complex clinical behavior with a wide array of determinants [43]. Adherence to HAART has increasingly been recognized as a major factor influencing biological and therapeutic outcome [44–46], as well as a powerful independent predictor of virologic suppression [47]. Lapses in adherence can lead to an increase in virus load and subsequent selection and transmission of drug-resistant strains of HIV to sex and drug-use partners.

Wilson et al. [48] examined adherence to ART and its association with risk behaviors among 766 women, and they found that lower rates of adherence were associated with younger age, active drug use, detectable virus load, and lower quality of life. Most notable was the association found between lower adherence rates and an increased risk of inconsistent condom use among sexually active women. Thus, adherence to ART is of particular concern when considered in conjunction with unprotected sexual behavior. Clinician-supported interventions to maintain and improve high levels of adherence to therapy must be an integral part of both patient care and HIV prevention.

**Screening, diagnosis, and treatment of STDs.** The population of patients at risk for HIV infection is also at risk for other STDs. Ulcerative and nonulcerative STDs may actually promote HIV transmission by increasing susceptibility to HIV infection in the seronegative population and by augmenting HIV infectiousness in the seropositive population. It is estimated that individuals who are infected with STDs are at least 2–5 times more likely than uninfected individuals to acquire HIV infection if they are exposed to the virus via sexual contact [49]. STDs that cause genital ulcers (e.g., syphilis, herpes, and chancroid) create breaks in the genital tract lining or skin, creating a portal of entry for HIV. Nonulcerative STDs (e.g., chlamydia, gonorrhea, and trichomoniasis) may also increase susceptibility by increasing the concentration of cells in genital secretions that can serve as targets for HIV (i.e., macrophages and CD4 cells). In addition, HIV-infected individuals who are coinfected with other STDs are more likely to have higher virus titers in their genital secretions, contributing further to a greater risk of transmitting HIV infection to their sex partners.

In terms of prevention of HIV infection, it has been shown at both the individual level and community level that treatment of STDs can lead to a decrease in the rates of HIV transmission. At the individual level, studies have shown that treatment of STDs in HIV-infected individuals decreases both the amount and frequency of HIV shedding. Thus, clinicians may have an important opportunity to reduce HIV transmission among their patients by aggressive and systematic screening and treatment of coexisting STDs. At the community level, there is evidence to support this as well. The Mwanza [50] and Rakai [51] studies from Africa evaluated the impact that treatment of STDs would have on the incidence of HIV transmission. The results of these 2 trials indicate that ongoing interventions to improve access to effective STD treatment services among symptomatic individuals reduces HIV transmission and is likely to be more effective than are intermittent interventions through such strategies as periodic mass treatment.
Public Health Initiative

Collaboration with existing public health efforts remains a critical aspect for clinicians in the multifactorial approach to prevention of HIV infection. These efforts include distribution of condoms and clean needles; use of HIV prevention posters, fact sheets, and brochures, which can be distributed in the clinics; programs to assist patients with partner counseling and notification; and referral services beyond the scope of the clinical setting for treatment of substance abuse, psychiatric treatment, and other risk behavior services.

CONCLUSIONS

The clinical care setting is an ideal place for the implementation of prevention practices, given the chronic nature of HIV disease, its requirement for multiple clinic visits over time, and the resultant close patient-provider relationship. Both behavioral and traditional medical interventions should become more formalized and incorporated into our clinical practice in a simple and efficient manner. Behavioral counseling that is supportive, interactive, directed toward a goal, and individualized should be administered at every clinical encounter, with scripted phrases used as needed. HAART and PEP should be administered as recommended by current guidelines, with the goal of both treatment of HIV disease as well as potential prevention of further spread of the disease to uninfected persons.

Promotion of adherence, risk reduction counseling, STD screening, diagnosis, and treatment should all be routine and integral components of patient care and, thus, clinicians’ prevention practice. The addition of behavioral counseling and medical HIV transmission prevention interventions in the clinical care setting to the current public health prevention effort is the next logical step in our prevention efforts and our attempt to curb the HIV epidemic.

References

25. Shain RN, Piper JM, Newton ER, et al. A randomized, controlled trial...


Early in the epidemic, HIV infection and AIDS were diagnosed for relatively few women and female adolescents (although we know now that many women were infected with HIV through injection drug use but that their infections were not diagnosed) [1]. Today, women account for more than one quarter of all new HIV/AIDS diagnoses. Women of color are especially affected by HIV infection and AIDS. In 2004 (the most recent year for which data are available), HIV infection was

- the leading cause of death for black women (including African American women) aged 25–34 years
- the 3rd leading cause of death for black women aged 35–44 years
- the 4th leading cause of death for black women aged 45–54 years
- the 4th leading cause of death for Hispanic women aged 35–44.

In the same year, HIV infection was the 5th leading cause of death among all women aged 35–44 years and the 6th leading cause of death among all women aged 25–34 years. The only diseases causing more deaths of women were cancer and heart disease [2].

- High-risk heterosexual contact was the source of 80% of these newly diagnosed infections [3].
- Women accounted for 26% of the estimated 37,163 diagnoses for adults and adolescents [3].
- Of the 126,964 women living with HIV/AIDS, 64% were black, 19% were white, 15% were Hispanic, 1% were Asian or Pacific Islander, and less than 1% were American Indian or Alaska Native [3].
- The number of HIV/AIDS diagnoses among female adults or adolescents decreased from 11,941 in 2001 to 9,708 in 2005 [3].
- According to a recent CDC study of more than 19,500 patients with HIV in 10 US cities, women were slightly less likely than men to receive prescriptions for the most effective treatments for HIV infection [4].

**STATISTICS**

**HIV/AIDS in 2005** *(The following bullets, except for the last one, are based on data from 33 states with long-term, confidential name-based HIV reporting.)*

- HIV/AIDS was diagnosed for an estimated 9,708 women [3].

*For a list of the 33 states, please refer to the box before the References.*
HIV/AIDS AMONG WOMEN

Transmission categories and race/ethnicity of women living with HIV/AIDS at the end of 2005

Note. Based on data from 33 states with long-term, confidential name-based HIV reporting.

AIDS in 2005

• Of 40,608 AIDS diagnoses in the 50 states and the District of Columbia, 10,774 (27%) were for women [3].

• The rate of AIDS diagnosis for black women (45.5/100,000 women) was approximately 23 times the rate for white women (2.0/100,000) and 4 times the rate for Hispanic women (11.2/100,000) [3].

• An estimated 95,959 women were living with AIDS, representing 23% of the estimated 421,873 people living with AIDS in the 50 states and the District of Columbia [3].

• An estimated 4,128 women with AIDS died, representing 25% of the 16,316 persons with AIDS who died in the 50 states and the District of Columbia [3].

• From the beginning of the epidemic (1981) through 2005, women accounted for 181,802 diagnoses, a number that represents 19% of the 952,629 AIDS diagnoses in the 50 states and the District of Columbia during this period [3].

• From the beginning of the epidemic through 2005, an estimated 85,844 women with AIDS died, accounting for 16% of the 530,756 persons with AIDS who died in the 50 states and the District of Columbia [3].

• Women with AIDS made up an increasing part of the epidemic. In 1992, women accounted for an estimated 14% of adults and adolescents living with AIDS in the 50 states and the District of Columbia [5]. By the end of 2005, this proportion had grown to 23% [3].

• Data from the 2005 census show that together, African American and Hispanic women represent 24% of all US women [6]. However, women in these 2 groups accounted for 82% (8,807/10,774) of the estimated total of AIDS diagnoses for women in 2005 [3].
HIV/AIDS AMONG WOMEN

RISK FACTORS AND BARRIERS TO PREVENTION

Younger Age

For women of all races and ethnicities, the largest number of HIV/AIDS diagnoses during recent years was for women aged 15–39. From 2001 through 2004, the number of HIV/AIDS diagnoses for women aged 15–39 decreased for white, black, and Hispanic women. There was an increase in the number of HIV/AIDS diagnoses during this period for Asian and Pacific Islander women and for American Indian and Alaska Native women aged 15–39 [7].

Diagnosis of HIV/AIDS in females aged 15-39 years

<table>
<thead>
<tr>
<th>Race/Ethnicity</th>
<th>2001 No. (%)</th>
<th>2004 No. (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>White</td>
<td>1,218 (63)</td>
<td>996 (56)</td>
</tr>
<tr>
<td>Black</td>
<td>5,229 (62)</td>
<td>4,091 (58)</td>
</tr>
<tr>
<td>Hispanic</td>
<td>1,192 (60)</td>
<td>819 (57)</td>
</tr>
</tbody>
</table>

Lack of Recognition of Partner’s Risk Factors

Some women may be unaware of their male partner’s risk factors for HIV infection (such as unprotected sex with multiple partners, sex with men, or injection drug use) [8]. Men who engage in sex both with men and women can acquire HIV from a male partner and then transmit the virus to female partners. In a 2003 report of a study of HIV-infected people (5,156 men and 3,139 women), 34% of black men who have sex with men (MSM), 26% of Hispanic MSM, and 13% of white MSM reported having had sex with women [9]. However, these women may not have known of their male partner’s bisexual activity: only 14% of white women, 6% of black women, and 6% of Hispanic women in this study acknowledged having a bisexual partner. In another CDC survey, 65% of the young men who had ever had sex with men also reported sex with women [10]. Women who have sex only with women and who have no other risk factors, such as injection drug use, are at very low risk for HIV infection (CDC, unpublished data, 2006).

High-Risk Heterosexual Risk Factors

Most women are infected with HIV through high-risk heterosexual contact [3]. Black and Hispanic women account for 81% of the women living with HIV/AIDS in 2005 who acquired HIV through high-risk heterosexual contact [3]. Lack of HIV knowledge, lower perception of risk,
HIV/AIDS among Women

Drug or alcohol use, and different interpretations of safer sex may contribute to this disproportion [11]. Relationship dynamics also play a role. For example, some women may not insist on condom use because they fear that their partner will physically abuse them or leave them [12]. Such sexual inequality is a major issue in relationships between young women and older men. In a CDC study of urban high schools, more than one third of black and Hispanic women had their first sexual encounter with a male who was older (3 or more years) [13]. These young women, compared with peers whose partners had been approximately their own age, had been younger at first sexual intercourse, less likely to have used a condom during first and most recently reported intercourse, or less likely to have used condoms consistently.

Socioeconomic Issues

Nearly 1 in 4 African Americans and 1 in 5 Hispanics live in poverty [20]. Socioeconomic problems associated with poverty, including limited access to high-quality health care; the exchange of sex for drugs, money, or to meet other needs; and higher levels of substance use can directly or indirectly increase HIV risk factors [21]. A study of HIV transmission among black women in North Carolina found that women with a diagnosis of HIV infection were significantly more likely than women who were not infected to be unemployed; to have had more sex partners; to use crack/cocaine; to exchange sex for money, shelter, or drugs; or to receive public assistance [22].

Racial/Ethnic Differences

The rates of HIV diagnosis and the risk factors for HIV infection differ for women of various races or ethnicities—a situation that must be considered when creating prevention programs. For example, even though the annual estimated rate of HIV diagnosis for black women decreased significantly—from 82.7 per 100,000 population in 2001 to 60.2 per 100,000 population in 2005—it remained 20 times the rate for white women [3, 23]. Overall, the rates of HIV diagnosis are much higher for black and Hispanic women than for white, Asian and Pacific Islander, or American Indian and Alaska Native women. The rates for black women are higher than the rates for all men except for black men [3, 24, 25].

Multiple Risk Factors

Some women infected with HIV report more than 1 risk factor, highlighting the overlap in risk factors such as inequality in relationships, socioeconomic stresses, substance abuse, and psychological issues. For example, in the North Carolina study of HIV infection in black women, the participants most commonly reported that their reasons for risky behavior were financial dependence on male partners, feeling invincible, low self-esteem coupled with the need to feel loved by a male figure, and alcohol and drug use [22].

Biologic Vulnerability and Sexually Transmitted Diseases

A woman is significantly more likely than a man to contract HIV infection during vaginal intercourse [14, 15]. Additionally, the presence of some sexually transmitted diseases greatly increases the likelihood of acquiring or transmitting HIV infection [16]. The rates of gonorrhea and syphilis are higher among women of color than among white women. These higher rates are especially marked at younger ages (15–24 years) [17].

Substance Use

An estimated 1 in 5 new HIV diagnoses for women are related to injection drug use [3]. Sharing injection equipment contaminated with HIV is not the only risk associated with substance use. Women who use crack cocaine or other noninjection drugs may also be at high risk for the sexual transmission of HIV if they sell or trade sex for drugs [18]. Also, both casual and chronic substance users are more likely to engage in high-risk behaviors, such as unprotected sex, when they are under the influence of drugs or alcohol [19].
PREVENTION

CDC estimates that 56,300 new HIV infections occurred in the United States in 2006 [26]. Populations of minority races/ethnicities are disproportionately affected by the HIV epidemic. To further reduce the incidence of HIV infection, CDC announced a new initiative, Advancing HIV Prevention, in 2003. This initiative comprises 4 strategies: making HIV testing a routine part of medical care, implementing new models for diagnosing HIV infections outside medical settings, preventing new infections by working with HIV-infected persons and their partners, and further decreasing perinatal HIV transmission.

In the United States, women, particularly women of color, are at risk for HIV infection. CDC, through the Department of Health and Human Services Minority AIDS Initiative, explores ways to reduce disparities in communities made up of persons of minority races/ethnicities who are at high risk for HIV infection. CDC is also conducting demonstration projects in which women’s social networks are used to reach high-risk persons in communities of color; CDC is also conducting outreach and testing for partners of HIV-infected men. Additionally, CDC recognizes the importance of further incorporating culture- and gender-relevant material into current interventions [27].

CDC funds prevention programs in state and local health departments and community-based organizations. The following are examples.

- In Illinois, Access Community Health Network, which is the largest network of community health centers in the nation, receives funding to implement counseling, testing, and referral (CTR) in Chicago communities with the highest rates of HIV diagnosis and funding to implement SISTA (Sisters Informing Sisters about Topics on AIDS), a social-skills training program aimed at reducing HIV sexual risk behavior among African American women at high risk for HIV infection.

- In Massachusetts, CAB Health & Recovery Services, Inc., receives funding for HIV risk-reduction counseling and prevention case management and for Women RISE (Risk Identification, Strategies, and Empowerment), an HIV prevention services program that engages women and their partners who are at very high risk for HIV infection, who are homeless and living in family shelters, or who are identified through street outreach.

- In California, the Orange County Bar Foundation adapts SISTA for Latinas aged 18–24 years.

- In Florida, the Center for Multicultural Wellness & Prevention, Inc., addresses, through SISTA and CTR, the health issues that affect African American and Haitian women.

- In New York, the Community Healthcare Network provides prevention services through counseling, comprehensive risk counseling and referral, and RAPP (Real AIDS Prevention Project) interventions to African American and Hispanic women.

CDC also funds research on interventions to reduce HIV-related risk behaviors or their outcomes. For example, the Women and Infants Demonstration Projects were focused on low-income, inner-city sexually active women to measure injection drug use, sexual behaviors, and rates of HIV testing, as well as sexually transmitted diseases and pregnancy. The demonstration projects increased condom use and resulted in the RAPP intervention package, which is available, along with training and technical assistance, from CDC.

CDC is actively involved in the promising area of microbicides—creams or gels that can be applied vaginally before sexual contact to prevent HIV transmission. The development of a safe, easy-to-use microbicide would be a milestone in the worldwide fight against HIV/AIDS. CDC is
supporting the search for an effective microbicide agent through several lines of research, including

- conducting laboratory and animal studies that can help evaluate the safety and the efficacy of microbicides before they are studied in humans.
- supporting clinical trials to assess the safety of microbicides in humans in the United States, Asia, and Africa. Current human clinical studies include a phase I safety trial of UC-781, which is being conducted among women in the United States and Thailand.

To reduce mother-to-child HIV transmission in the United States, CDC has distributed approximately $10 million annually since 1999 to several national organizations and a number of states with high HIV/AIDS rates. These funds support perinatal HIV prevention programs, enhanced surveillance for HIV-infected mothers and babies, education, and capacity building among health care providers and public health practitioners.

REFERENCES


Understanding HIV and AIDS Data

AIDS surveillance: Through a uniform system, CDC receives reports of AIDS cases from all US states and dependent areas. Since the beginning of the epidemic, these data have been used to monitor trends because they are representative of all areas. The data are statistically adjusted for reporting delays and for the redistribution of cases initially reported without risk factors. As treatment has become more available, trends in new AIDS diagnoses no longer accurately represent trends in new HIV infections; these data now represent persons who are tested late in the course of HIV infection, who have limited access to care, or in whom treatment has failed.

HIV surveillance: Monitoring trends in the HIV epidemic today requires the collection of information on HIV cases that have not progressed to AIDS. Areas with requirements for confidential name-based HIV infection reporting use the same uniform system for data collection on HIV cases as for AIDS cases. A total of 33 states (Alabama, Alaska, Arizona, Arkansas, Colorado, Florida, Idaho, Indiana, Iowa, Kansas, Louisiana, Michigan, Minnesota, Mississippi, Missouri, Nebraska, Nevada, New Jersey, New Mexico, New York, North Carolina, North Dakota, Ohio, Oklahoma, South Carolina, South Dakota, Tennessee, Texas, Utah, Virginia, West Virginia, Wisconsin, and Wyoming) have collected these data for at least 5 years, providing sufficient data to monitor HIV trends.

HIV/AIDS: This term is used to refer to 3 categories of diagnoses collectively: (1) a diagnosis of HIV infection (not AIDS), (2) a diagnosis of HIV infection and a later diagnosis of AIDS, (3) concurrent diagnoses of HIV infection and AIDS.


For more information . . .

CDC HIV/AIDS
http://www.cdc.gov/hiv
CDC HIV/AIDS resources

CDC-INFO
1-800-232-4636
Information about personal risk and where to get an HIV test

CDC National HIV Testing Resources
http://www.hivtest.org
Location of HIV testing sites

CDC National Prevention Information Network (NPIN)
1-800-458-5231
http://www.cdcnpin.org
CDC resources, technical assistance, and publications

AIDSinfo
1-800-448-0440
http://www.aidsinfo.nih.gov
Resources on HIV/AIDS treatment and clinical trials
The HIV/AIDS epidemic is a serious threat to the Hispanic/Latino community. In 2005, HIV/AIDS was the fourth leading cause of death among Hispanic/Latino men and women aged 35 to 44 [1].

THE NUMBERS

HIV/AIDS in 2006

• Hispanics/Latinos accounted for 18% of the 35,314 new HIV/AIDS diagnoses in 33 states with long-term, confidential name-based HIV reporting [2].

• Hispanics/Latinos accounted for 17% of the 491,727 persons (including children) living with HIV/AIDS in the 33 states [2].

• For Hispanic/Latino men living with HIV/AIDS, the most common methods of HIV transmission were (in order) sexual contact with other men, injection drug use, and high-risk heterosexual contact. For Hispanic/Latina women living with HIV/AIDS, the most common methods of transmission were high-risk heterosexual contact and injection drug use [2].

Race/ethnicity of persons (including children) with HIV/AIDS diagnosed during 2006

AIDS in 2006

• Hispanics/Latinos accounted for 19% of new AIDS diagnoses and 19% of all people living with AIDS in the 50 states and the District of Columbia [2].

• Of the rates of AIDS diagnoses for adults and adolescents in all racial and ethnic groups, the second highest (after the rate for blacks) was the rate for Hispanics/Latinos [2].

• Although Hispanics/Latinos made up only about 13% of the population of the United States, they accounted for 16% of the estimated 982,498 AIDS cases diagnosed in the 50 states and the District of Columbia since the beginning of the epidemic [2].

• By the end of 2006, an estimated 80,690 Hispanics/Latinos with AIDS in the 50 states and the District of Columbia had died [2].

PREVENTION CHALLENGES

A number of cultural, socioeconomic, and health-related factors contribute to the HIV epidemic and prevention challenges in the US Hispanic/Latino community.

• Behavioral risk factors for HIV infection differ by country of birth. For example, data suggest that Hispanics/Latinos born in Puerto Rico are more likely than other Hispanics/Latinos to contract HIV as a result of injection drug use or high-risk heterosexual contact. By contrast, sexual contact with other men is the primary cause of HIV infections among Hispanic/Latino men born in Central or South America, Cuba, Mexico, or the United States [2].

• Hispanic/Latina women and Hispanic/Latino men are most likely to be infected with HIV as a result of sex with men [2]. Therefore, prevention program staff need to address issues specific to the Hispanics/Latinos to whom a particular program is directed: for example, condom usage (men and women) or the balance of power within relationships (especially women).

• Injection drug use continues to be a risk factor for Hispanics/Latinos, particularly those living in Puerto Rico [2]. Both casual and chronic substance users are
more likely to engage in risky sexual behaviors, such as unprotected sex, when they are under the influence of drugs or alcohol.

- The rates of sexually transmitted diseases, which can increase the chances of contracting HIV, are higher for Hispanics/Latinos. In 2006, the rate of chlamydial infection for Hispanics/Latinos was about 3 times the rate for whites (not Hispanic/Latino), and the rates of gonorrhea and syphilis for Hispanics/Latinos were about twice the rates for whites [3].

- Certain cultural beliefs can affect one’s risk for HIV infection. For example, among men, machismo has positive implications for HIV prevention, such as strength and protection of the family; however, proving masculinity through power and dominance can lead both straight and gay Hispanic/Latino men to engage in risky sexual behavior.

- Greater acculturation into the US culture has both negative (engaging in behaviors that increase the risk for HIV infection) and positive (communicating with partners about practicing safer sex) effects on the health behaviors of Hispanics/Latinos.

- More than 1 in 5 (21.9%) Hispanics/Latinos live in poverty [4]. Problems associated with poverty, including unemployment, a lack of formal education, inadequate health insurance, and limited access to high-quality health care, can increase the risk for HIV infection.

- The migration patterns, social structure, language barriers, and lack of regular health care among transient Hispanic/Latino immigrants can affect awareness and hinder access to HIV/AIDS prevention and care.

WHAT CDC IS DOING

To reduce the incidence of HIV infection, CDC released Revised Recommendations for HIV Testing of Adults, Adolescents, and Pregnant Women in Health-Care Settings in 2006. These new recommendations advise routine HIV screening for adults, adolescents, and pregnant women in health care settings in the United States. CDC also

- Conducts epidemiologic and behavioral research focused on Hispanics/Latinos
- Supports efforts to reduce the health disparities experienced in the communities of minority races and ethnicities at high risk for HIV infection
- Provides effective, scientifically based interventions to organizations serving Hispanics/Latinos and is tailoring other effective behavioral interventions to Hispanics/Latinos who are at high risk for HIV infection
- Builds the capacity of programs that serve Hispanics/Latinos through partnerships with national, regional, and nongovernmental organizations

In 2006, CDC provided 56 awards to community-based organizations in the United States and Puerto Rico that focus primarily on Hispanics/Latinos. CDC also provides funding through state, territorial, and local health departments to organizations serving this population. In addition, CDC provides training for researchers of minority races/ethnicities and in 2002 established the Minority HIV/AIDS Research Initiative (MARI) to create partnerships between CDC epidemiologists and researchers who are members of minority races and ethnicities and who work in communities of color. CDC invests $2 million per year in the program and since 2003 has funded 13 junior investigators at 12 sites across the country.
HIV/AIDS Among Hispanics --- United States, 2001--2005

In the United States, Hispanics are affected disproportionately by human immunodeficiency virus (HIV) infection and acquired immunodeficiency syndrome (AIDS). Although Hispanics accounted for 14.4% of the U.S. population in 2005 (1), they accounted for 18.9% of persons who received an AIDS diagnosis (2). The rate of HIV diagnosis among Hispanics also remains disproportionately high; in 2005, the annual rate of HIV diagnosis for Hispanics was three times that for non-Hispanic whites. To better characterize HIV infection and AIDS among Hispanics in the United States, CDC analyzed selected characteristics of Hispanics in whom HIV infection was diagnosed during 2001--2005 and those living with AIDS in 2005. The results indicated that the mode of HIV infection for Hispanics varied by place of birth, suggesting that all HIV-prevention measures might not be equally effective among Hispanics and that HIV educational activities should address cultural and behavioral differences among Hispanic subgroups.

This analysis includes cases of HIV/AIDS diagnosed among Hispanic adults and adolescents aged ≥13 years during 2001--2005 in 33 states and cases of Hispanics living with HIV or AIDS in 50 states and the District of Columbia in 2005. Included are HIV cases reported to CDC from the 33 states* that have conducted name-based HIV reporting since at least 2001. Confidential name-based HIV and AIDS reporting has achieved high levels of accuracy and reliability (CDC, unpublished data, 2005). HIV/AIDS cases include those with 1) a diagnosis of HIV infection that have not progressed to AIDS, 2) a diagnosis of HIV infection followed by a diagnosis of AIDS, 3) and concurrent diagnoses of AIDS and HIV infection (i.e., in the same month).

Cases were classified according to the following transmission categories: 1) male-to-male sexual contact (i.e., among men who have sex with men [MSM]); 2) injection-drug use (IDU); 3) MSM with IDU; 4) high-risk heterosexual contact (i.e., with a person of the opposite sex known to be HIV infected or at high risk for HIV/AIDS [e.g., MSM or injection-drug user]); and 5) other modes of infection (e.g., receipt of transfusion of blood, blood components, or tissue transplant) and unknown risk factors. Cases reported with unknown risk factors were reclassified into transmission categories (e.g., MSM, IDU, MSM and IDU, high-risk heterosexual contact, and other) in accordance with methods described previously (3). Potential duplicate cases were identified based on unique identifiers and selected demographic characteristics and were eliminated on both state and national levels.

For 2005, annual HIV/AIDS diagnosis rates per 100,000 population were calculated for Hispanics, non-Hispanic whites, and non-Hispanic blacks. Data were adjusted for reporting delays† (3). The number of Hispanics living with HIV or AIDS at the end of 2005 was calculated based on reported cases adjusted for delays in reporting and deaths; this calculation does not account for undiagnosed cases.

During 2001--2005, a total of 184,167 adults and adolescents had HIV/AIDS diagnosed in the 33 states and reported to CDC. Of these, 33,398 (18%) were Hispanics; 93,017 (51%) were non-Hispanic blacks; 54,029 (29%) were non-Hispanic whites; 1% were Asian/Pacific Islanders; and <1% were American Indian/Alaska Natives. The mode of HIV infection for 61% of Hispanic males was male-to-male sexual contact, 17% of infections occurred through high-risk heterosexual contact, and 17% occurred through IDU. Among Hispanic females with HIV/AIDS diagnoses, 76% were exposed through high-risk heterosexual contact, suggesting that all HIV-prevention measures might not be equally effective among Hispanic females and that HIV educational activities should address cultural and behavioral differences among Hispanic subgroups.
contact, and 23\% were exposed through IDU (Table 1).

In 2005, the overall annual rate of HIV/AIDS diagnosis among Hispanic males was 56.2 per 100,000 population and among Hispanic females was 15.8 per 100,000 population. For Hispanic males, the highest rate of HIV diagnosis (86.3 per 100,000) occurred among those aged 30--39 years; for Hispanic females, the highest rate (25.0 per 100,000) occurred among those aged 40--49 years. The overall rates for non-Hispanic white and non-Hispanic black males in 2005 were 18.2 and 124.8, respectively, and the rates for non-Hispanic white and non-Hispanic black females were 3.0 and 60.2, respectively.

The mode of HIV infection among Hispanics varied by place of birth (Table 2). Infection through male-to-male sexual contact was more common among Hispanics born in South America (65\%), Cuba (62\%), and Mexico (54\%) than among Hispanics born in the United States (46\%). A greater proportion of Hispanics born in the Dominican Republic (47\%) and Central America (45\%) were infected through high-risk heterosexual contact, compared with Hispanics born in the United States (28\%). Hispanics born in Puerto Rico had a greater proportion of HIV infections attributed to IDU (33\%) than those born in the United States (22\%).

In 2005, in the 33 states, the rate of living with HIV infection among Hispanics was estimated at 173.0 per 100,000 population (Table 3). Estimated HIV prevalence among Hispanics ranged from 34.3 per 100,000 population in Wyoming to 443.0 in New York. In the 50 states and DC, the rate of living with AIDS among Hispanics was estimated at 244.2 per 100,000 population. Estimated AIDS prevalence ranged from 28.7 per 100,000 population in Montana to 1,165.8 per 100,000 population in DC.

Reported by: L Espinoza, DDS, KL Dominguez, MD, RA Romaguera, DMD, X Hu, LA Valleroy, PhD, HI Hall, PhD, Div of HIV/AIDS Prevention, National Center for HIV/AIDS, Viral Hepatitis, STD and TB Prevention, CDC.

Editorial Note:

These results confirm a previous report of disproportionate rates of HIV diagnosis among Hispanics, who have the second highest rate among all racial/ethnic groups in the United States (4). During 2001--2004, HIV-diagnosis rates among Hispanics declined by 4.7\% and 13.0\% among Hispanic males and females, respectively (4). These decreases among Hispanics might have resulted from decreased incidence of HIV infection (e.g., in response to prevention measures) or a decrease in HIV testing among Hispanics. However, this report indicates that Hispanics are not a homogenous group, and risk factors differ for Hispanic subpopulations.

Nearly half of U.S. Hispanics in whom HIV infection was diagnosed were not born in the United States. Hispanics born in Mexico and elsewhere often migrate to the United States to work as laborers and in service occupations. Migration might contribute to an increase in HIV risk behaviors, perhaps because change in residence can be followed by homelessness, loneliness, isolation, separation from usual sex partners, and financial instability. These factors can be associated with new sex partners, illegal drug use, and inadequate access to health information and health-care services (5).

During 2001--2005, the primary mode of HIV infection among Hispanic males was male-to-male sexual contact. A recent study of HIV risk behaviors among MSM reported that Hispanic and non-Hispanic black MSM were more likely than non-Hispanic white MSM to report inconsistent condom use during anal sex (6). However, male-to-male sexual contact is not the most common transmission category for Hispanics for certain places of birth. High-risk heterosexual contact was more common among Hispanics born in Central America and the Dominican Republic than Hispanics born in South America, Cuba, Mexico, Puerto Rico, and the United States. In addition, HIV knowledge and perceptions of risk differ among U.S. Hispanic subgroups. Immigrants born in Cuba, Mexico, and Puerto Rico who were injection-drug users reported less AIDS knowledge than U.S.-born injection-drug users (7).
The finding that a greater proportion of Puerto Rico-born Hispanics residing in the 33 states are infected through IDU is consistent with previous reports (8) and might be the result of both greater prevalence of IDU and increased levels of high-risk behaviors related to IDU (e.g., frequency of injecting and sharing syringes) compared with other Hispanics (9). U.S. Hispanic subgroups of varied national origin or ancestry differ in IDU-related behaviors. Puerto Rico-born injection-drug users are more likely to share syringes, cotton, or rinse water and to inject more frequently than Puerto Ricans born in the United States (10).

The findings in this report are subject to at least four limitations. First, although AIDS is a reportable condition in all 50 states, name-based HIV data were available from only 33 states. These states represented an estimated 63% of all AIDS cases and 56% of AIDS cases among Hispanics in the United States during 2001--2005. The exclusion (2) of data from some states with high AIDS prevalence and a large Hispanic population (e.g., California) results in an underrepresentation of cases among Hispanics. Second, the assumptions by which the approximately 32% of cases that had no known risk factors were redistributed among transmission categories might no longer be valid; these assumptions are being reevaluated. Third, misclassification of Hispanics as members of other races/ethnicities or inability to include undocumented migrant workers might have resulted in underestimations of the number of Hispanics overall and in Hispanic subgroups. Finally, birthplace information was missing for approximately 24% of Hispanics in this analysis. Depending on the distribution of birthplaces for persons with missing information, transmission-category prevalences for certain subgroups might have been larger or smaller.

The disproportionate rate of HIV infection among Hispanics might reflect the failure of HIV-prevention programs to reach Hispanics at high risk for acquiring or transmitting HIV infection. More specifically, the difference in HIV transmission categories among Hispanics by place of birth might represent differences in acculturation, linguistic ability, socioeconomic status, and stigma associated with homosexuality or male-to-male sex. CDC recently established an internal committee to develop a National Plan of Action to reduce the number of new HIV infections among Hispanics and to increase access to culturally appropriate prevention, care, and treatment services. The plan is aimed at enhancing research, policy, and community involvement to increase capacity to deliver appropriate HIV-prevention services to Hispanics. CDC will expand its partnerships with other federal agencies, state and local health departments, academic institutions, and community-based organizations to identify specific steps to implement the National Plan of Action. Because the Hispanic population in the United States is expected to nearly triple between 2000 and 2050, additional attention to the impact of HIV on this population is warranted.

References


† Reporting delays (i.e., time between diagnosis and report) can differ by geographic location, age, sex, transmission category, and race/ethnicity. Adjustments for reporting time were calculated for HIV and AIDS cases using a maximum likelihood statistical procedure that accounts for differences in reporting time for the preceding characteristics while assuming the reporting delay has remained constant over time. Adjustments also were made based on the redistribution of cases across transmission categories by sex, race/ethnicity, and geographic region for cases diagnosed 3--10 years earlier and initially classified as reported with unknown risk factors but later reclassified.


Table 1

<table>
<thead>
<tr>
<th>Age group (yrs)</th>
<th>Total Males</th>
<th>Total Females</th>
<th>Total</th>
</tr>
</thead>
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<tr>
<td>13--19</td>
<td>520 (2)</td>
<td>269 (3)</td>
<td>790 (2)</td>
</tr>
<tr>
<td>20--29</td>
<td>6,084 (23)</td>
<td>1,745 (23)</td>
<td>7,829 (23)</td>
</tr>
<tr>
<td>30--39</td>
<td>9,797 (38)</td>
<td>2,438 (32)</td>
<td>12,235 (37)</td>
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<tr>
<td>40--49</td>
<td>6,332 (24)</td>
<td>1,983 (26)</td>
<td>8,315 (25)</td>
</tr>
<tr>
<td>50--59</td>
<td>2,215 (9)</td>
<td>841 (11)</td>
<td>3,056 (9)</td>
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<tr>
<td>≥60</td>
<td>879 (3)</td>
<td>205 (4)</td>
<td>1,084 (3)</td>
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</table>

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<th>Transmission category</th>
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<th>Total Females</th>
<th>Total</th>
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<td>15,742 (61)</td>
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<td>15,742 (47)</td>
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<tr>
<td>Injection-drug use</td>
<td>4,472 (17)</td>
<td>1,737 (23)</td>
<td>6,209 (18)</td>
</tr>
<tr>
<td>Male-to-male sexual contact and injection-drug use</td>
<td>1,184 (4)</td>
<td>—</td>
<td>1,184 (3)</td>
</tr>
<tr>
<td>High-risk heterosexual contact</td>
<td>4,301 (17)</td>
<td>5,728 (76)</td>
<td>10,029 (30)</td>
</tr>
<tr>
<td>Other§§</td>
<td>120 (1)</td>
<td>106 (1)</td>
<td>226 (1)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Area of residence††</th>
<th>Total Males</th>
<th>Total Females</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rural</td>
<td>1,173 (4)</td>
<td>304 (4)</td>
<td>1,477 (4)</td>
</tr>
<tr>
<td>Suburban</td>
<td>1,961 (8)</td>
<td>523 (7)</td>
<td>2,484 (7)</td>
</tr>
<tr>
<td>Urban</td>
<td>22,156 (86)</td>
<td>6,620 (87)</td>
<td>28,776 (86)</td>
</tr>
<tr>
<td>Unknown</td>
<td>538 (2)</td>
<td>124 (2)</td>
<td>662 (2)</td>
</tr>
</tbody>
</table>

* All estimates have been adjusted for reporting delays and the reclassification of cases with unknown risk factors for HIV infection.
† Data were reported by 33 U.S. states with confidential, name-based reporting; Alabama, Alaska, Arizona, Arkansas, Colorado, Florida, Idaho, Indiana, Iowa, Kansas, Louisiana, Michigan, Minnesota, Mississippi, Missouri, Nebraska, Nevada, New Jersey, New Mexico, New York, North Carolina, North Dakota, Ohio, Oklahoma, South Carolina, South Dakota, Tennessee, Texas, Utah, Virginia, West Virginia, Wisconsin, and Wyoming.
§§ Because column totals were calculated independently of the values for the subpopulations, the values in each column might not sum to the column total.
†† Includes receipt of transfusion of blood, blood components, or tissue and unknown risk factor.
* Rural: Nonmetropolitan area. Suburban: 50,000--500,000 population. Urban: >500,000 population.

Table 2
### TABLE 2. Estimated number and percentage of HIV/AIDS diagnoses among Hispanic adults and adolescents aged ≥13 years, by transmission category and place of birth — 33 states,† 2001–2005

<table>
<thead>
<tr>
<th>Place of birth</th>
<th>Male-to-male sexual contact</th>
<th>Male-drug use</th>
<th>Male</th>
<th>Female</th>
<th>Male-to-male sexual contact and injection-drug use</th>
<th>High-risk heterosexual contact</th>
<th>Other§ Total**</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No. (%)</td>
<td>No. (%)</td>
<td>No. (%)</td>
<td>No. (%)</td>
<td>No. (%)</td>
<td>No. (%)</td>
<td>No. (%)</td>
</tr>
<tr>
<td>United States</td>
<td>6,190 (46)</td>
<td>2,001 (15)</td>
<td>922 (7)</td>
<td>553 (4)</td>
<td>1,400 (10)</td>
<td>2,985 (18)</td>
<td>75 (1)</td>
</tr>
<tr>
<td>Central America</td>
<td>657 (41)</td>
<td>139 (8)</td>
<td>43 (3)</td>
<td>39 (2)</td>
<td>338 (21)</td>
<td>393 (24)</td>
<td>23 (1)</td>
</tr>
<tr>
<td>South America</td>
<td>1,330 (65)</td>
<td>107 (5)</td>
<td>40 (2)</td>
<td>45 (2)</td>
<td>225 (11)</td>
<td>286 (14)</td>
<td>10 (1)</td>
</tr>
<tr>
<td>Cuba</td>
<td>732 (62)</td>
<td>76 (6)</td>
<td>18 (2)</td>
<td>50 (4)</td>
<td>185 (16)</td>
<td>111 (9)</td>
<td>3 (&lt;1)</td>
</tr>
<tr>
<td>Dominican Republic</td>
<td>181 (30)</td>
<td>90 (13)</td>
<td>40 (7)</td>
<td>15 (2)</td>
<td>106 (17)</td>
<td>185 (30)</td>
<td>6 (1)</td>
</tr>
<tr>
<td>Mexico</td>
<td>2,163 (54)</td>
<td>962 (9)</td>
<td>64 (2)</td>
<td>153 (4)</td>
<td>656 (16)</td>
<td>577 (14)</td>
<td>42 (1)</td>
</tr>
<tr>
<td>Puerto Rico</td>
<td>602 (29)</td>
<td>502 (25)</td>
<td>161 (8)</td>
<td>89 (4)</td>
<td>243 (12)</td>
<td>421 (21)</td>
<td>15 (1)</td>
</tr>
<tr>
<td>Other†</td>
<td>177 (40)</td>
<td>62 (14)</td>
<td>28 (6)</td>
<td>13 (3)</td>
<td>77 (17)</td>
<td>78 (18)</td>
<td>3 (1)</td>
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<tr>
<td>Unknown</td>
<td>3,710 (47)</td>
<td>1,142 (14)</td>
<td>421 (5)</td>
<td>227 (3)</td>
<td>1,060 (13)</td>
<td>1,302 (16)</td>
<td>57 (1)</td>
</tr>
<tr>
<td>Total**</td>
<td>15,742 (47)</td>
<td>4,472 (13)</td>
<td>1,737 (5)</td>
<td>1,184 (4)</td>
<td>4,300 (13)</td>
<td>5,728 (17)</td>
<td>235 (1)</td>
</tr>
</tbody>
</table>

* All estimates have been adjusted for reporting delays and the recategorization of cases with unknown risk factors for HIV infection.

† Data were reported by 33 U.S. states with confidential, name-based reporting: Alabama, Alaska, Arizona, Arkansas, Colorado, Florida, Idaho, Indiana, Iowa, Kansas, Louisiana, Michigan, Minnesota, Mississippi, Missouri, Nebraska, Nevada, New Jersey, New Mexico, New York, North Carolina, North Dakota, Ohio, Oklahoma, South Carolina, South Dakota, Tennessee, Texas, Utah, Virginia, West Virginia, Wisconsin, and Wyoming.

‡ Heterosexual contact with a sex partner known to have HIV infection or to be at high risk for HIV infection.

§ Includes receipt of transfusion of blood, blood components, or tissue and unknown risk factor.

** Because row and column totals were calculated independently of the values for the subpopulations, the values in each row and column might not sum to the row or column total.

†† Places of birth other than those specified.

### Table 3

<table>
<thead>
<tr>
<th>Area of residence</th>
<th>Living with HIV infection (not AIDS)§</th>
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</thead>
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<tr>
<td></td>
<td>No.</td>
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<td>76</td>
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<td>Alaska</td>
<td>16</td>
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<td>Arizona</td>
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<tr>
<td>Arkansas</td>
<td>49</td>
<td>52.2</td>
</tr>
<tr>
<td>California</td>
<td>384</td>
<td>125.2</td>
</tr>
<tr>
<td>Colorado</td>
<td>17,270</td>
<td>184.5</td>
</tr>
<tr>
<td>Connecticut</td>
<td>2,147</td>
<td>740.6</td>
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<tr>
<td>Delaware</td>
<td>438</td>
<td>1,165.8</td>
</tr>
<tr>
<td>District of Columbia</td>
<td>7,992</td>
<td>286.9</td>
</tr>
<tr>
<td>Florida</td>
<td>6,184</td>
<td>222.0</td>
</tr>
<tr>
<td>Georgia</td>
<td>100</td>
<td>140.4</td>
</tr>
<tr>
<td>Hawaii</td>
<td>2,410</td>
<td>179.7</td>
</tr>
<tr>
<td>Idaho</td>
<td>207</td>
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<tr>
<td>Illinois</td>
<td>71</td>
<td>89.4</td>
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<tr>
<td>Indiana</td>
<td>132</td>
<td>79.6</td>
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<tr>
<td>Iowa</td>
<td>103</td>
<td>166.7</td>
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<tr>
<td>Kansas</td>
<td>202</td>
<td>193.9</td>
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<tr>
<td>Kentucky</td>
<td>397</td>
<td>164.4</td>
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<td>201</td>
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<tr>
<td>Maine</td>
<td>205</td>
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<tr>
<td>Maryland</td>
<td>66</td>
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<td>Michigan</td>
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<td>87.1</td>
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<td>Minnesota</td>
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<tr>
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<tr>
<td>Missouri</td>
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<td>183.0</td>
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<tr>
<td>State</td>
<td>2001</td>
<td>2002</td>
</tr>
<tr>
<td>-------------</td>
<td>------</td>
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</tr>
<tr>
<td>Montana</td>
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<tr>
<td>Nebraska</td>
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<td>3,095</td>
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<td>361</td>
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<td>10,781</td>
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<td>408</td>
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<tr>
<td>South Carolina</td>
<td>142</td>
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<td>6</td>
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<tr>
<td>Tennessee</td>
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<td>Texas</td>
<td>5,267</td>
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<td>Utah</td>
<td>137</td>
<td>71.8</td>
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<tr>
<td>Vermont</td>
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<tr>
<td>Virginia</td>
<td>496</td>
<td>145.4</td>
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<tr>
<td>Washington</td>
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<tr>
<td>West Virginia</td>
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<td>124.3</td>
</tr>
<tr>
<td>Wyoming</td>
<td>9</td>
<td>34.3</td>
</tr>
</tbody>
</table>

Total** | 31,851 | 187.0 | 173.0 | 77,817 | 244.2 |

* Rates are per 100,000 population. All estimates have been adjusted for reporting delays.


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**Questions or messages regarding errors in formatting should be addressed to mmwrq@cdc.gov.**