IMMUNE DISORDERS

CCV

Microbiology

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HYPERSENSITIVITIES

- Hypersensitivity
  - Any immune response against a foreign antigen exaggerated beyond the norm
- Four types
  - Type I (immediate)
  - Type II (cytotoxic)
  - Type III (immune complex–mediated)
  - Type IV (delayed or cell-mediated)

2

HYPERSENSITIVITIES

- **Type I (Immediate) Hypersensitivity**
  - Localized or systemic reaction that results from the release of inflammatory molecules in response to an antigen
  - Develops within seconds or minutes following exposure to an antigen
  - Commonly called *allergies*
  - The antigens that stimulate it are called *allergens*

3

FIGURE 18.1A THE MECHANISMS OF A TYPE I HYPERSENSITIVITY REACTION.

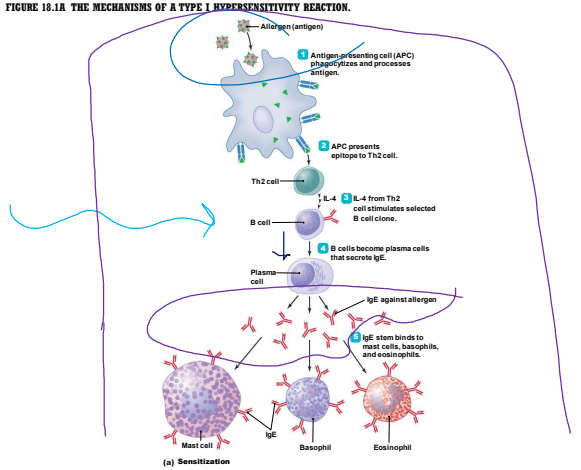

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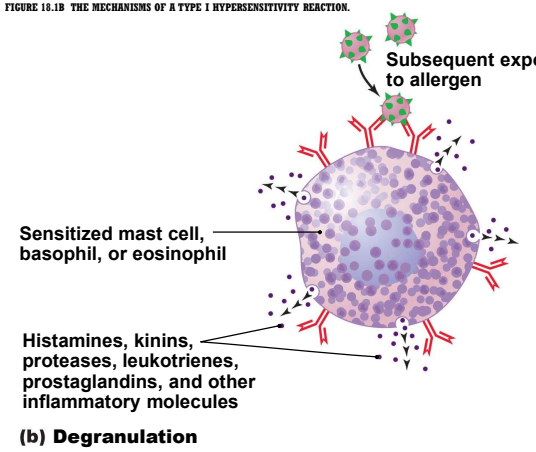
FIGURE 18.1B THE MECHANISMS OF A TYPE I HYPERSENSITIVITY REACTION.



(b) Degranulation

5

HYPERSENSITIVITIES

- **Type I (Immediate) Hypersensitivity**
  - The roles of degranulating cells in an allergic reaction
    - Mast cells
      - Distributed throughout the body in connective tissue
      - Have granules that contain inflammatory chemicals
        - Degranulation releases histamine, kinins, proteases, leukotrienes, and prostaglandins

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TABLE 18.1 Inflammatory Molecules Released from Mast Cells

Molecules	Role in Hypersensitivity Reactions
Released During Degranulation	
Histamine	Causes smooth muscle contraction, increased vascular permeability, and irritation
Kinins	Cause smooth muscle contraction, inflammation, and irritation
Proteases	Damage tissues and activate complement
Synthesized in Response to Inflammation	
Leukotrienes	Cause slow, prolonged smooth muscle contraction, inflammation, and increased vascular permeability
Prostaglandins	Some contract smooth muscle; others relax it

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HYPERSENSITIVITIES

- **Type I (Immediate) Hypersensitivity**
  - The roles of degranulating cells in an allergic reaction
    - Basophils
      - Least numerous leukocyte in the blood
      - Have granules that contain inflammatory chemicals
      - Degranulate like mast cells when encounter allergens

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HYPERSENSITIVITIES

- **Type I (Immediate) Hypersensitivity**
  - The roles of degranulating cells in an allergic reaction
    - Eosinophils
      - Eosinophilia is the accumulation of eosinophils in blood
      - Mast cell degranulation can trigger the release of eosinophils from the bone marrow
      - Eosinophils in the bloodstream can degranulate
        - Release large amounts of leukotrienes
          - Increases severity of a hypersensitivity response

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HYPERSENSITIVITIES

- **Type I (Immediate) Hypersensitivity**
  - Clinical signs of localized allergic reactions
    - Usually mild and localized
    - Site of reaction depends on portal of entry
    - Inhaled allergens may cause hay fever
    - Small inhaled allergens may reach lungs and cause asthma
    - Some allergens may cause inflammation of the skin, called *hives* or *urticaria*

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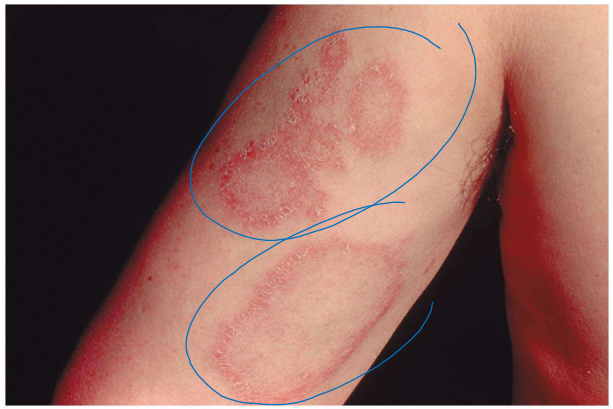
FIGURE 18.3 URTICARIA.



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HYPERSENSITIVITIES

- **Type I (Immediate) Hypersensitivity**
  - Clinical signs of systemic allergic reactions
    - Many mast cells may degranulate at once
      - Release large amounts of histamine and inflammatory mediators
    - Acute anaphylaxis or anaphylactic shock can result
    - Clinical signs are those of suffocation
    - Must be treated promptly with epinephrine
    - Common causes include bee stings and certain foods

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HYPERSENSITIVITIES

- **Type I (Immediate) Hypersensitivity**
  - Diagnosis of type I hypersensitivity
    - Based on detection of high levels of allergen-specific IgE
    - Test referred to as ImmunoCAP specific IgE blood test, CAP RAST, or Pharmacia CAP
    - Can also diagnose using skin tests

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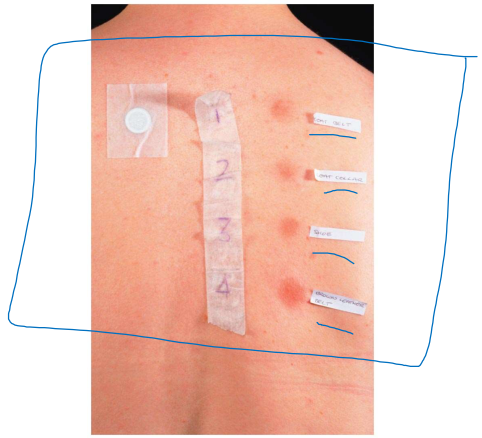
FIGURE 18.4 SKIN TESTS FOR DIAGNOSING TYPE I HYPERSENSITIVITY.



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HYPERSENSITIVITIES

- **Type I (Immediate) Hypersensitivity**
  - Prevention of type I hypersensitivity
    - Identification and avoidance of allergens
    - Food allergens are identified using an elimination diet
    - Immunotherapy ("allergy shots") can help prevent allergic reactions
      - Administration of a series of injections of dilute allergen
      - Must be repeated every two to three years
      - Not effective in treating asthma

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HYPERSENSITIVITIES

- **Type I (Immediate) Hypersensitivity**
  - Treatment of type I hypersensitivity
    - Administer drugs that counteract inflammatory mediators
      - Antihistamines neutralize histamine
    - Treat asthma with a glucocorticoid and a bronchodilator
    - Epinephrine neutralizes many mechanisms of anaphylaxis
      - Relaxes smooth muscle
      - Reduces vascular permeability
      - Used in emergency treatment of severe asthma and anaphylactic shock

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HYPERSENSITIVITIES

- **Type II (Cytotoxic) Hypersensitivity**
  - Results when cells are destroyed by an immune response
    - Often the combined activities of complement and antibodies
  - A component of many autoimmune diseases
  - Two significant examples
    - Destruction of blood cells following an incompatible blood transfusion
    - Destruction of fetal red blood cells

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HYPERSENSITIVITIES

- **Type II (Cytotoxic) Hypersensitivity**
  - The ABO system and transfusion reactions
    - Blood group antigens are surface molecules of red blood cells
    - Each person's red blood cells have A antigen, B antigen, both antigens, or neither antigen
    - Transfusion reaction can result if an individual receives different blood type
    - Donor's blood group antigens may stimulate the production of antibodies in the recipient that destroy the transfused cells

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HYPERSENSITIVITIES

- **Type II (Cytotoxic) Hypersensitivity**
  - The ABO system and transfusion reactions
    - Recipient has preexisting antibodies to foreign blood group antigens
      - Immediate destruction of donated blood cells can occur
    - Recipient has no preexisting antibodies to foreign blood group antigens
      - Transfused cells initially circulate and function normally
      - Eventually recipient's immune system mounts a primary response against the foreign antigens and destroys them

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FIGURE 18.5 EVENTS LEADING TO HEMOLYSIS.

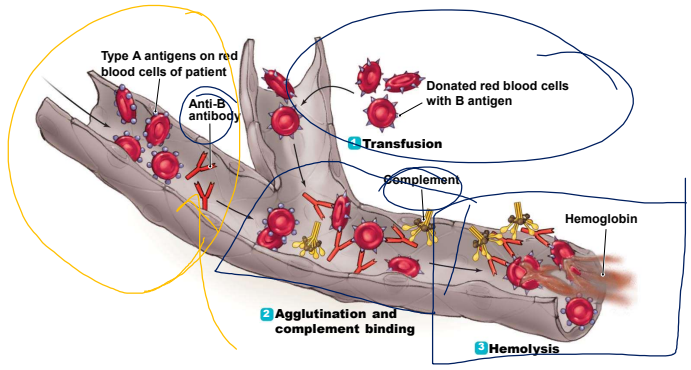

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TABLE 18.2 ABO Blood Group Characteristics and Donor/Recipient Matches

ABO Blood Group	ABO Antigen(s) Present	Antibodies Present	Can Donate To	Can Receive From
A	A	Anti-B	A or AB	A or O
B	B	Anti-A	B or AB	B or O
AB	A and B	None	AB	A, B, AB, or O (universal recipient)
O	None	Both anti-A and anti-B	A, B, AB, or O (universal donor)	O

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HYPERSENSITIVITIES

D-antigen/Rh

- **Type II (Cytotoxic) Hypersensitivity**
  - The Rh system and hemolytic disease of the newborn
    - Rh antigen
      - Common to red blood cells of humans and rhesus monkeys
    - About 85% of humans are Rh-positive (Rh+)
    - If Rh− woman is carrying an Rh+ fetus, the fetus may be at risk for hemolytic disease
    - Administration of anti-Rh immunoglobulin, called RhoGAM, has reduced cases of hemolytic disease of the newborn

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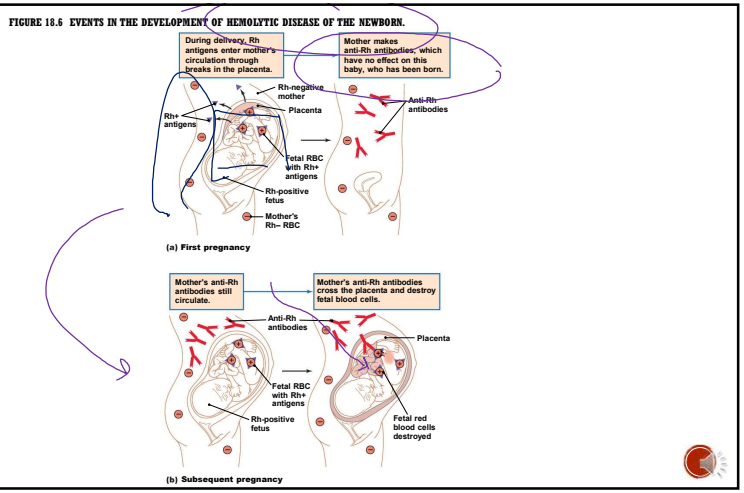
FIGURE 18.6 EVENTS IN THE DEVELOPMENT OF HEMOLYTIC DISEASE OF THE NEWBORN.



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HYPERSENSITIVITIES

- **Type II (Cytotoxic) Hypersensitivity**
  - Drug-induced cytotoxic reactions
    - Some drugs are too small to trigger immune responses
      - May become antigenic if bind to larger molecules
    - Can produce various diseases
      - Immune thrombocytopenic purpura
      - Agranulocytosis
      - Hemolytic anemia

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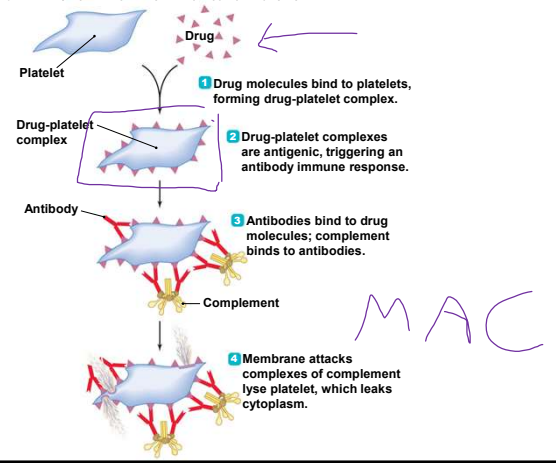
FIGURE 18.7 EVENTS IN THE DEVELOPMENT OF IMMUNE THROMBOCYTOPENIC PURPURA.



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HYPERSENSITIVITIES

- **Type III (Immune Complex–Mediated) Hypersensitivity**
  - Caused by formation of immune complexes
    - Triggers release of inflammatory chemicals
  - Can cause localized reactions
    - Hypersensitivity pneumonitis
    - Glomerulonephritis
  - Can cause systemic reactions
    - Systemic lupus erythematosus
    - Rheumatoid arthritis

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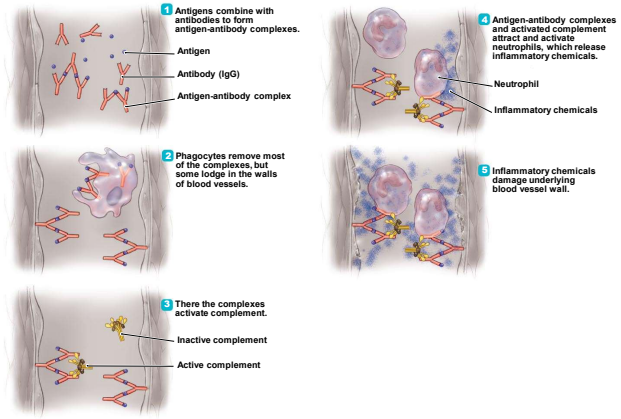
FIGURE 18.8 THE MECHANISM OF TYPE III (IMMUNE COMPLEX-MEDIATED) HYPERSENSITIVITY.



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HYPERSENSITIVITIES

- **Type III (Immune Complex–Mediated) Hypersensitivity**
  - Hypersensitivity pneumonitis
    - Inhalation of antigens deep in the lungs stimulates the production of antibodies
    - Subsequent inhalation of the same antigen stimulates the formation of immune complexes
      - Activates complement

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HYPERSENSITIVITIES

- **Type III (Immune Complex–Mediated) Hypersensitivity**
  - Glomerulonephritis
    - Immune complexes circulating in the bloodstream are deposited in the walls of glomeruli (small blood vessels in the kidneys)
    - Damage to the glomerular cells impedes blood filtration
    - Kidney failure and ultimately death result

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HYPERSENSITIVITIES

- **Type III (Immune Complex–Mediated) Hypersensitivity**
  - Rheumatoid arthritis (RA)
    - Immune complexes are deposited in the joint
      - Results in release of inflammatory chemicals
      - The joints begin to break down and become distorted
        - Damage is progressively more severe
    - Trigger is not well understood
    - Treated with anti-inflammatory drugs

biologia

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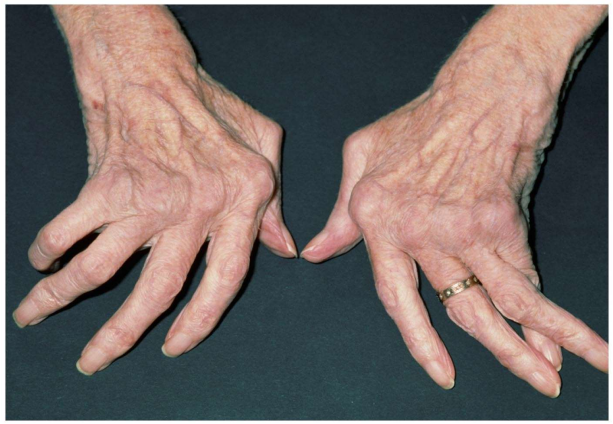
FIGURE 18.9 THE CRIPPLING DISTORTION OF JOINTS CHARACTERISTIC OF RHEUMATOID ARTHRITIS.



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AUTOIMMUNE DISEASES

- **Type III (Immune Complex–Mediated) Hypersensitivity**
  - Systemic lupus erythematosus (SLE)
    - Autoantibodies against DNA cause immune complex formation
      - Can cause glomerulonephritis and kidney failure
    - Many other autoantibodies can also occur
      - Against red blood cells, platelets, lymphocytes, muscle cells
    - Trigger unknown
    - Treatment with immunosuppressive drugs reduces autoantibody formation
    - Treatment with corticosteroids reduces inflammation

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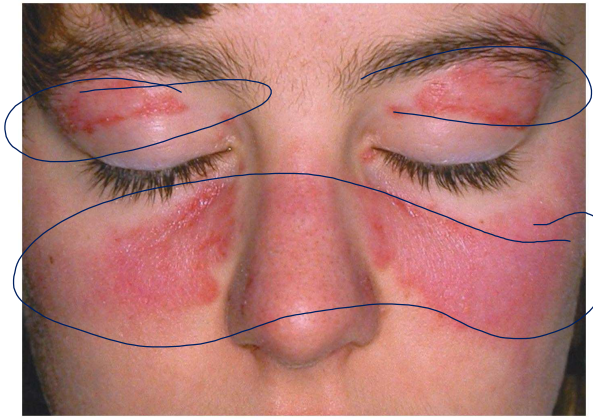
FIGURE 18.10 THE CHARACTERISTIC FACIAL RASH OF SYSTEMIC LUPUS ERYTHEMATOSUS.



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HYPERSENSITIVITIES

- **Type IV (Delayed or Cell-Mediated) Hypersensitivity**
  - Inflammation occurs 12 to 24 hours after contact with certain antigens
  - Results from the actions of antigen, antigen-presenting cells, and T cells
  - Delay reflects the time it takes for macrophages and T cells to migrate to and proliferate at the site of the antigen

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HYPERSENSITIVITIES

- **Type IV (Delayed or Cell-Mediated) Hypersensitivity**
  - The tuberculin response
    - Skin exposed to tuberculosis or tuberculosis vaccine reacts to an injection of tuberculin beneath the skin
    - Used to diagnose contact with antigens of *M. tuberculosis*
      - No response when individual has not been infected or vaccinated
      - Red, hard swelling develops in individuals previously infected or immunized
    - Response mediated by memory T cells, causing a slowly developing inflammation

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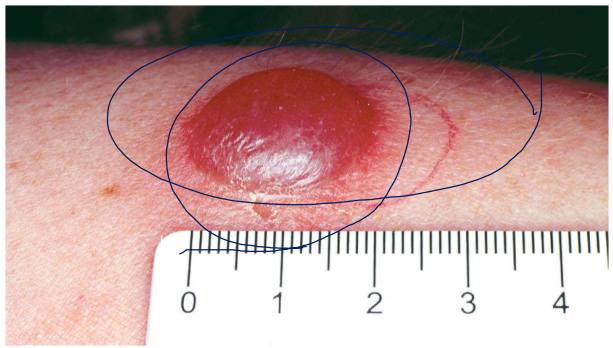
FIGURE 18.11 A POSITIVE TUBERCULIN TEST, A TYPE IV HYPERSENSITIVITY RESPONSE.



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HYPERSENSITIVITIES

- **Type IV (Delayed or Cell-Mediated) Hypersensitivity**
  - Allergic contact dermatitis
    - Cell-mediated immune response resulting in an intensely irritating skin rash
    - Triggered by chemically modified skin proteins that the body regards as foreign
    - In severe cases, acellular, fluid-filled blisters develop
    - Can be caused by poison ivy, formaldehyde, cosmetics, and chemicals used to produce latex
    - Treated with corticosteroids

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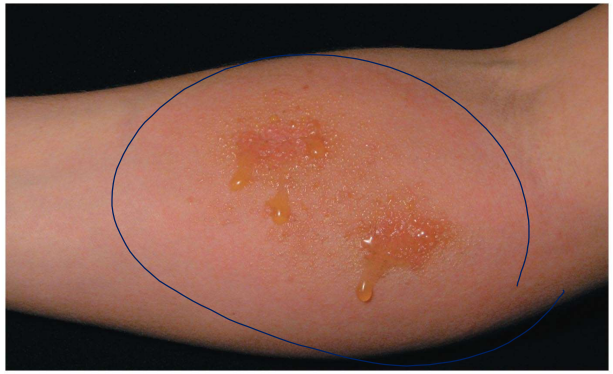
FIGURE 18.12 ALLERGIC CONTACT DERMATITIS, A TYPE IV HYPERSENSITIVITY RESPONSE.



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HYPERSENSITIVITIES

- **Type IV (Delayed or Cell-Mediated) Hypersensitivity**
  - Graft rejection
    - Rejection of tissues or organs that have been transplanted
    - Grafts perceived as foreign by a recipient undergo rejection
    - Normal immune response against foreign MHC proteins present on graft cells
    - Likelihood of rejection depends on the degree to which the graft is foreign to the recipient
      - Based on the type of graft

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HYPERSENSITIVITIES

- **Type IV (Delayed or Cell-Mediated) Hypersensitivity**
  - Graft-versus-host disease
    - Donated bone marrow cells regard the patient's cells as foreign
    - Donor and recipient differ in MHC class I molecules
      - Grafted T cells attack all of the recipient's tissues
    - Donor and recipient differ in MHC class II molecules
      - Grafted T cells attack the host's antigen-presenting cells
    - Immunosuppressive drugs can stop graft-versus-host disease

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HYPERSENSITIVITIES

- **Type IV (Delayed or Cell-Mediated) Hypersensitivity**
  - Donor-recipient matching and tissue typing
    - MHC compatibility between donor and recipient is difficult because of high degree of variability
      - The more closely the donor and recipient are related, the smaller the difference in their MHC
    - Preferable that grafts be donated by a parent or sibling
    - Tissue typing is used to match donor and recipient

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TABLE 18.3 The Characteristics of the Four Types of Hypersensitivity Reactions

Descriptive	Name	Cause	Time Course	Characteristic Cells Involved
Type I	Immediate hypersensitivity	Antibody (IgE) on sensitized cells' membranes binds antigen, causing degranulation	Seconds to minutes	Mast cells, basophils, and eosinophils
Type II	Cytotoxic hypersensitivity	Antibodies and complement lyse target cells	Minutes to hours	Red blood cells
Type III	Immune complex–mediated hypersensitivity	Nonphagocytized complexes of antibodies and antigens trigger mast cell degranulation	Several hours	Neutrophils
Type IV	Delayed hypersensitivity	T cells attack the body's cells	Several days	Activated T cells

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HYPERSENSITIVITIES

- **Type IV (Delayed or Cell-Mediated) Hypersensitivity**
  - The actions of immunosuppressive drugs
    - Immunosuppressive drugs are important to success of modern transplantation
    - Important classes of immunosuppressive drugs
      - Glucocorticoids
      - Cytotoxic drugs
      - Cyclosporine
      - Lymphocyte-depleting therapies

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TABLE 18.4 The Four Classes of Immunosuppressive Drugs

Class	Examples	Action
Glucocorticoids	Prednisone, methylprednisolone	Anti-inflammatory; kills T cells
Cytotoxic drugs	Cyclophosphamide, azathioprine, mycophenolate mofetil, brequinar sodium, leflunomide	Blocks cell division nonspecifically
Cyclosporine	Cyclosporine	Blocks T cell responses
Lymphocyte-depleting therapies	Antilymphocyte globulin, monoclonal antibodies	Kills T cells nonspecifically, kills activated T cells, inhibits IL-2 reception

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AUTOIMMUNE DISEASES

- **Causes of Autoimmune Diseases**
  - Occur more often in the elderly
  - Are more common in women than men
  - May result when an individual begins to make antibodies or cytotoxic T cells against normal body cells

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AUTOIMMUNE DISEASES

- **Causes of Autoimmune Diseases**
  - Estrogen may stimulate destruction of tissue by cytotoxic T cells
  - Some maternal cells may cross the placenta, colonize the fetus, and trigger autoimmune disease later in life
  - Fetal cells may cross the placenta and trigger autoimmunity in the mother
  - Environmental factors such as viral infections
  - Genetic factors such as certain MHC genes
  - T cells may encounter self-antigens that are normally "hidden"
  - Microorganisms may trigger autoimmunity due to molecular mimicry
  - Failure of the normal control mechanisms of the immune system

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AUTOIMMUNE DISEASES

- **Examples of Autoimmune Diseases**
  - Two major categories
    - Systemic autoimmune diseases
    - Single-organ autoimmune diseases
      - Can affect many different organs
        - Blood cells
        - Endocrine organs
        - Nervous tissue
        - Connective tissue

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IMMUNODEFICIENCY DISEASES

- Conditions resulting from defective immune mechanisms
- Two general types
  - Primary immunodeficiency diseases
    - Result from some genetic or developmental defect
    - Develop in infants and young children
  - Acquired immunodeficiency diseases
    - Develop as a direct consequence of some other recognized cause
    - Develop in later life

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TABLE 18.5 Some Primary Immunodeficiency Diseases

Disease	Defect	Manifestation
Chronic granulomatous disease	Ineffective phagocytes	Uncontrolled infections
Severe combined immunodeficiency disease (SCID)	A lack of T cells and B cells	No resistance to any type of infection, leading to rapid death
Bruton-type agammaglobulinemia	A lack of B cells and thus a lack of immunoglobulins	Death from overwhelming bacterial infections
DiGeorge syndrome	A lack of T cells and thus no cell-mediated immunity	Death from overwhelming viral infections

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IMMUNODEFICIENCY DISEASES

- **Acquired Immunodeficiency Diseases**
  - Result from a number of causes
    - Age: as we age, immune function decreases naturally, especially T-cell function
    - Severe stress
      - Suppression of cell-mediated immunity results from an excess production of corticosteroids
    - Malnutrition and environmental factors
      - Inhibit production of B cells and T cells
    - Acquired immunodeficiency syndrome (AIDS)
      - Opportunistic infections, low CD4 cells, presence of HIV

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IMMUNODEFICIENCY DISEASES

- **Acquired Immunodeficiency Diseases**
  - Signs and symptoms of AIDS
    - Not a disease but a syndrome
    - Syndrome = a group of signs, symptoms, and diseases associated with a common pathology
    - Defined as certain opportunistic or rare infections along with infection by HIV or a severe decrease in the number of helper T cells and a positive test for HIV
    - Several infections and diseases define AIDS

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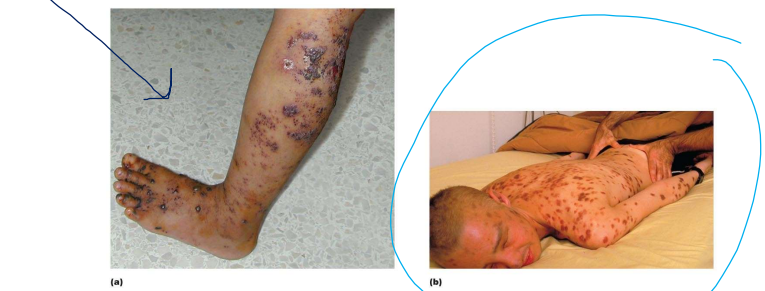
FIGURE 18.14 DISEASES ASSOCIATED WITH AIDS.



(a) (b)

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TABLE **18.6** Opportunistic Infections Associated with AIDS

Disease	Causative Agent	Organ Primarily Affected (Chapter Where Covered)
Coccidioidomycosis	*Coccidioides* (fungus)	Lung (22)
Cytomegalovirus disease	*Cytomegalovirus*	Brain (20), liver (23)
Diarrhea (severe and prolonged)	Various bacteria, *Cryptosporidium* (protozoan)	Intestines (23)
Herpes	*Herpesvirus*	Skin (19)
Histoplasmosis	*Histoplasma* (fungus)	Lung (22)
Kaposi's sarcoma	Human herpesvirus 8	Blood vessels (21)
Meningitis	*Cryptococcus* (yeast), *Listeria* (bacterium)	Brain and meninges (20)
Oral hairy leukoplakia	*Lymphocryptovirus* (Epstein-Barr virus)	Tongue (23)
Pneumonia	*Pneumocystis* (fungus)	Lung (22)
Shingles	*Varicellovirus*	Skin (19)
Thrush	*Candida* (yeast)	Mouth and tongue (23), vagina (24)
Toxoplasmosis	*Toxoplasma* (protozoan)	Lungs, liver, heart (21)
Tuberculosis	*Mycobacterium*	Lung (22)

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IMMUNODEFICIENCY DISEASES

- **Acquired Immunodeficiency Diseases**
  - AIDS pathogen and its virulence factors
    - HIV
      - Enveloped, +ssRNA virus
      - Retrovirus – uses reverse transcriptase to make DNA copy of genome
  - Two major types of HIV
    - HIV-1 is prevalent in the United States and Europe
    - HIV-2 is prevalent in West Africa

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IMMUNODEFICIENCY DISEASES

- **Acquired Immunodeficiency Diseases**
  - AIDS pathogen and its virulence factors
    - Structure of HIV
      - Viral envelope has two antigenic glycoproteins
        - gp120
          - Primary attachment molecule of HIV
          - Antigenic variability during prolonged infection
        - gp41
          - Promotes fusion of viral envelope to target cell
      - Viral characteristics impede immune clearance of HIV

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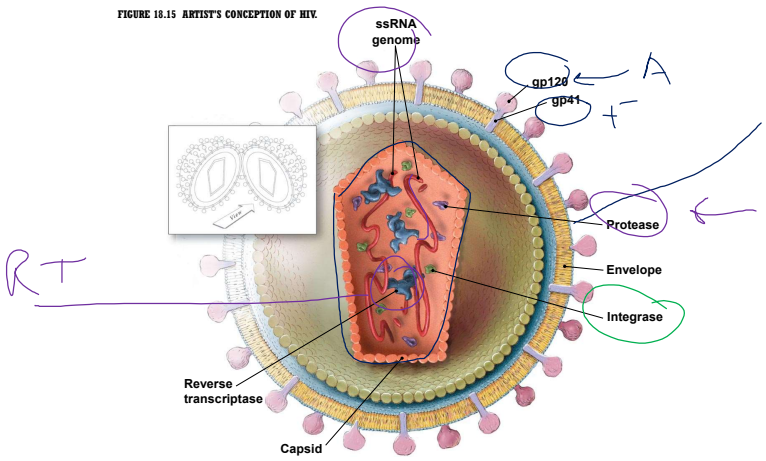
FIGURE 18.15 ARTIST'S CONCEPTION OF HIV.



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IMMUNODEFICIENCY DISEASES

- **Acquired Immunodeficiency Diseases**
  - AIDS pathogen and its virulence factors
    - Origin of HIV
      - Likely arose from mutation of simian immunodeficiency virus
      - May have emerged in the human population around 1930
      - Whether the two HIV types are derived from the same or different SIV strains is unknown

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FIGURE 18.16 THE REPLICATION CYCLE OF HIV.

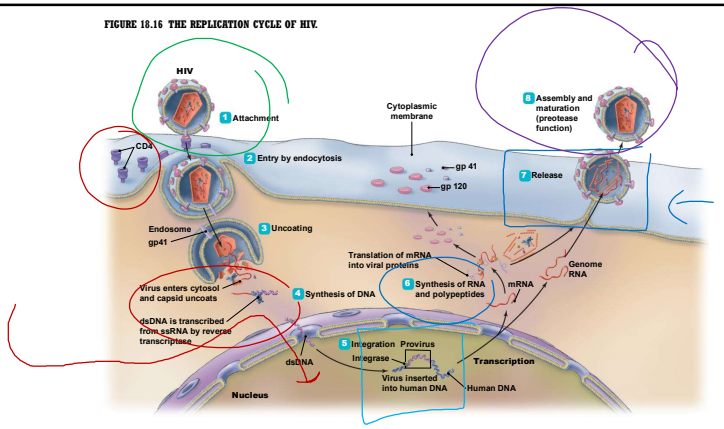

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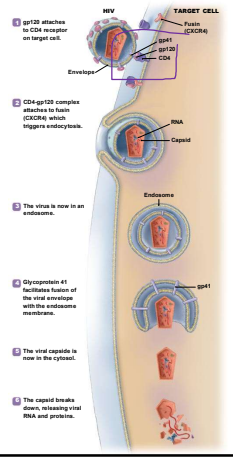
FIGURE 18.17 THE PROCESS BY WHICH HIV ATTACHES TO AND ENTERS A HOST CELL.



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IMMUNODEFICIENCY DISEASES

- **Acquired Immunodeficiency Diseases**
  - Details of release, assembly, and maturation
    - HIV exits cell at lipid rafts in the cytoplasmic membrane
      - Lipid raft components become the viral envelope
    - Capsomeres form immature capsid outside the cell
    - Viral protease releases proteins that produce a mature virus
      - Protease inhibitors are used to treat HIV

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FIGURE 18.18 ACTION OF REVERSE TRANSCRIPTASE, DEPICTED HERE AS THREE DISTINCT STEPS.

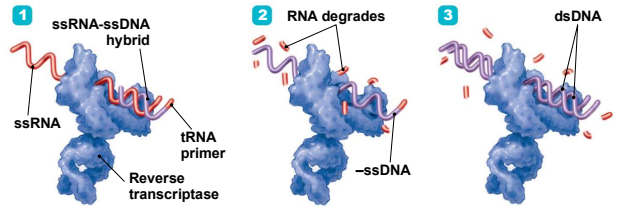

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TABLE 18.7 Characteristics of HIV That Challenge the Immune System

Characteristic	Effect(s)
Retrovirus with a genome that consists of two copies of +ssRNA	Reassortment of viral genes possible; reverse transcription produces much mutation and thus genetic variation; genome integrates into host's chromosome
Targets helper T cells especially, but also macrophages, dendritic cells, and muscle cells, and possibly liver, nerve, and epithelial cells	Permanently infects key cells of host's immune system
Antigenic variability	Numerous antigenic variations due to mutations helps virus evade host's immune response
Induces formation of syncytia	Increases routes of infection; intracellular site helps virus evade immune detection

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IMMUNODEFICIENCY DISEASES

- **Acquired Immunodeficiency Diseases**
  - Pathogenesis of AIDS
    - Only humans replicate HIV
    - HIV destroys the immune system
      - Destruction of helper T cells relates to course of AIDS

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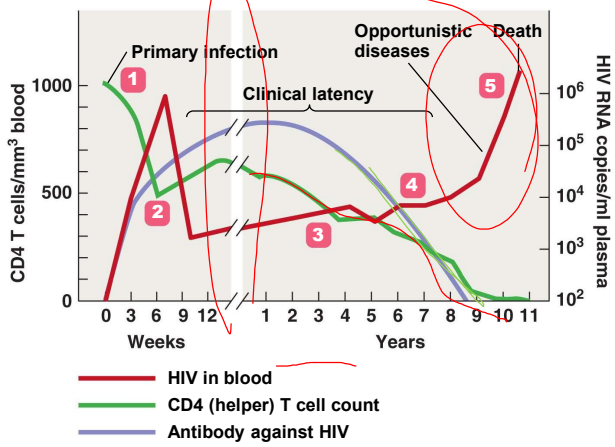
FIGURE 18.19 THE COURSE OF AIDS FOLLOWS THE COURSE OF HELPER T CELL DESTRUCTION.



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IMMUNODEFICIENCY DISEASES

- **Acquired Immunodeficiency Diseases**
  - Epidemiology of AIDS
    - AIDS was first recognized in young male homosexuals in the United States
    - AIDS is now found worldwide
    - HIV found in blood, semen, saliva, vaginal secretions, and breast milk can cause infections
      - Blood and semen are more infective than other secretions
      - Infected fluid must be injected or contact a tear or lesion in the skin or mucous membranes

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FIGURE 18.20 THE GLOBAL DISTRIBUTION OF HIV/AIDS.

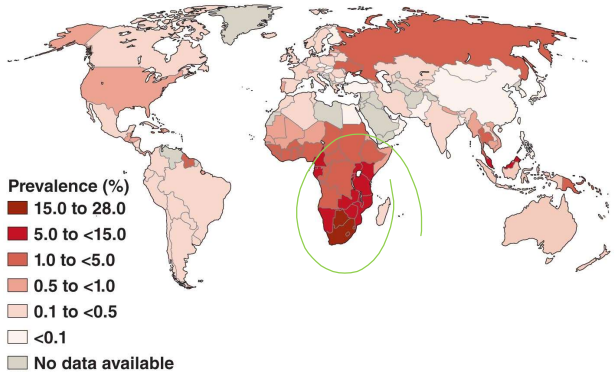

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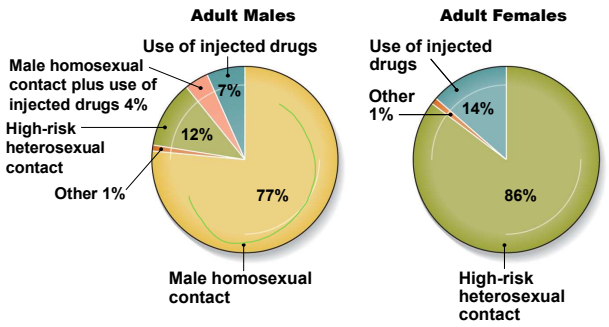
FIGURE 18.21 MODES OF HIV TRANSMISSION IN PEOPLE OVER 12 YEARS OF AGE IN THE UNITED STATES DURING 2010.



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IMMUNODEFICIENCY DISEASES

- **Acquired Immunodeficiency Diseases**
  - Epidemiology of AIDS
    - Some behaviors increase the risk of HIV infection
      - Anal intercourse
      - Sexual promiscuity
      - Intravenous drug use
      - Intercourse with someone in these categories
    - A few cases of casual HIV spread have been documented

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IMMUNODEFICIENCY DISEASES

- **Acquired Immunodeficiency Diseases**
  - Diagnosis, treatment, and prevention
    - Diagnosis
      - AIDS diagnosis is based on symptoms, low levels of CD4 lymphocytes, and presence of antibodies against HIV
      - Antibodies against HIV are detected by ELISA or western blot
        - Positive test does not indicate presence of AIDS
      - Long-term nonprogressors
        - Do not develop AIDS
        - Possible reasons: defective virions, poor binding of HIV to cells, or well-developed immune systems

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IMMUNODEFICIENCY DISEASES

- **Acquired Immunodeficiency Diseases**
  - Diagnosis, treatment, and prevention
    - Treatment
      - ART currently used to reduce viral replication
        - Cocktail of antiviral drugs
        - Does not cure the infection
          - Inhibits HIV replication
        - Patient can live relatively normal life while on treatment

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IMMUNODEFICIENCY DISEASES

- **Acquired Immunodeficiency Diseases**
  - Diagnosis, treatment, and prevention
    - Prevention
      - Behavioral changes can slow the AIDS epidemics
        - Abstinence and safe sex
        - Use of clean needles
        - Providing antiviral drugs to infected pregnant women
        - Screening of blood products
        - Use of protective wear to prevent contact with blood
        - Circumcision reduces infection through sexual activity

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IMMUNODEFICIENCY DISEASES

- **Acquired Immunodeficiency Diseases**
  - Diagnosis, treatment, and prevention
    - Prevention
      - Behavioral changes can slow the AIDS epidemics
        - Pre-exposure prophylaxis (oral tenofovir)
        - Vaginal application of tenofovir before and after intercourse reduces the chance of infection

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