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15-3 Incompatibility of blood group antigens causes type II hypersensitivity reactions

15-4 Hyperacute rejection of transplanted organs is a type II hypersensitivity reaction

15-5 Anti-HLA antibodies can arise from pregnancy, blood transfusion, or previous transplants

15-6 Transplant rejection and graft-versus-host disease are type IV hypersensitivity reactions

Summary

Transplantation of solid organs

15-7 Organ transplantation involves procedures that inflame the donated organ and the transplant recipient

15-8 Acute rejection is a type IV hypersensitivity caused by effector T cells responding to HLA differences between donor and recipient

15-9 HLA differences between transplant donor and recipient activate numerous alloreactive T cells

15-10 Chronic rejection of organ transplants is caused by a type III hypersensitivity reaction

15-11 Matching donor and recipient HLA class I and II allotypes improves the success of transplantation

15-12 Immunosuppressive drugs make allogeneic transplantation possible as routine therapy

15-13 Some treatments induce immunosuppression before transplantation

15-14 T-cell activation can be targeted by immunosuppressive drugs

15-15 Alloreactive T-cell co-stimulation can be blocked with a soluble form of CTLA4

15-16 Blocking cytokine signaling can prevent alloreactive T-cell activation

15-17 Cytotoxic drugs target the replication and proliferation of alloantigen-activated T cells

15-18 Patients needing a transplant outnumber the available organs

15-19 The need for HLA matching and immunosuppressive therapy varies with the organ transplanted

Summary

Hematopoietic cell transplantation

15-20 Hematopoietic cell transplantation is a treatment for genetic diseases of blood cells

15-21 Allogeneic hematopoietic cell transplantation is the preferred treatment for many cancers

15-22 After hematopoietic cell transplantation, the patient is attacked by alloreactive T cells in the graft

15-23 HLA matching of donor and recipient is most important for hematopoietic cell transplantation

15-24 Minor histocompatibility antigens trigger alloreactive T cells in recipients of HLA-identical transplants

15-25 Some GVHD helps engraftment and prevents relapse of malignant disease

15-26 NK cells also mediate graft-versus-leukemia effects

15-27 Hematopoietic cell transplantation can induce tolerance of a solid organ transplant

Summary

Summary to Chapter 15

Questions

Chapter 16

Disruption of Healthy Tissue by the Adaptive Immune Response

16-1 Every autoimmune disease resembles a type II, III, or IV hypersensitivity reaction

16-2 Autoimmune diseases arise when tolerance to self antigens is lost

16-3 HLA is the dominant genetic factor affecting susceptibility to autoimmune disease

16-4 HLA associations reflect the importance of T-cell tolerance in preventing autoimmunity

16-5 Binding of antibodies to cell-surface receptors causes several autoimmune diseases

16-6 Organized lymphoid tissue sometimes forms at sites inflamed by autoimmune disease

16-7 The antibody response to an autoantigen can broaden and strengthen by epitope spreading

16-8 Intermolecular epitope spreading occurs in systemic autoimmune disease

16-9 Intravenous immunoglobulin is a therapy for autoimmune diseases
16-10 Monoclonal antibodies that target TNF-α and B cells are used to treat rheumatoid arthritis

16-11 Rheumatoid arthritis is influenced by genetic and environmental factors

16-12 Autoimmune disease can be an adverse side-effect of an immune response to infection

16-13 Noninfectious environmental factors affect the development of autoimmune disease

16-14 Type 1 diabetes is caused by the selective destruction of insulin-producing cells in the pancreas

16-15 Combinations of HLA class II allotypes confer susceptibility and resistance to type 1 diabetes

16-16 Celiac disease is a hypersensitivity to food that has much in common with autoimmune disease

16-17 Celiac disease is caused by the selective destruction of intestinal epithelial cells

16-18 Senescence of the thymus and the T-cell population contributes to autoimmunity

16-19 Autoinflammatory diseases of innate immunity

Summary to Chapter 16

Questions

Chapter 17

Cancer and Its Interactions With the Immune System

17-1 Cancer results from mutations that cause uncontrolled cell growth

17-2 A cancer arises from a single cell that has accumulated multiple mutations

17-3 Exposure to chemicals, radiation, and viruses facilitates progression to cancer

17-4 Certain common features distinguish cancer cells from normal cells

17-5 Immune responses to cancer have similarities with those to virus-infected cells

17-6 Allogeneic differences in MHC class I molecules enable cytotoxic T cells to eliminate tumor cells

17-7 Mutations acquired by somatic cells during oncogenesis can give rise to tumor-specific antigens

17-8 Cancer/testis antigens are a prominent type of tumor-associated antigen

17-9 Successful tumors evade and manipulate the immune response

17-10 Vaccination against human papillomaviruses can prevent cervical and other genital cancers

17-11 Vaccination with tumor antigens can cause cancer to regress but it is unpredictable

17-12 Monoclonal antibodies that interfere with negative regulators of the immune response can be used to treat cancer

17-13 T-cell responses to tumor cells can be improved with chimeric antigen receptors

17-14 The antitumor response of γ:δ T cells and NK cells can be augmented

17-15 T-cell responses to tumors can be improved by adoptive transfer of antigen-activated dendritic cells

17-16 Monoclonal antibodies are valuable tools for the diagnosis of cancer

17-17 Monoclonal antibodies against cell-surface antigens are increasingly used in cancer therapy

Summary to Chapter 17

Questions