Robbins Review Questions - Chapter 6

1. Major components of innate immunity include all of the following except
    a. Epithelial barriers
    b. Phagocytic cells
    c. Dendritic cells
    d. Lymphocytes
2. Natural killer cells are part _____ (innate/adaptive) immunity?
3. Antibodies are part of _____ (innate/adaptive) immunity?
4. Briefly explain the significance of pattern recognition receptors and specifically Toll-like receptors (TLRs).
5. Which of the following provides defense against intracellular microbes?
    a. Humoral immunity
    b. Innate immunity
    c. Cell-mediated immunity
    d. B lymphocytes
6. What type of immunity is responsible for protecting against extracellular microbes and their toxins?
7. Which cells are the only immune cells capable of producing antibodies?
8. These cells are the primary antigen-presenting cells responsible for initiating T-cell responses against protein antigens.
    a. B cells
    b. T cells
    c. Follicular cells
    d. Dendritic cells
9. The function of _____ is to destroy irreversibly stressed and abnormal cells, such as virus-infected cells and tumor cells.
    a. NK cells
    b. B cells
    c. T cells
    d. Langerhans cells
10. The principal generative lymphoid organs are the _____ and the _____.
    a. Thymus and spleen
    b. Thymus and bone marrow
    c. Bone marrow and lymph nodes
    d. Mucosal associated lymphoid tissues (MALT) and lymph nodes
11. Which of the following are a peripheral lymphoid organ/tissue?
    a. Thymus
    b. Bone marrow
    c. Spleen
    d. None of the above
12. Follicles are discrete structures found within the periphery/cortex of lymph nodes and contain concentrations of
    a. T cells
    b. B cells
    c. NK cells

    d. Dendritic cells
13. Briefly explain the function of MHC molecules.
14. Which cells express Class 1 MHC molecules?
15. Class 1 MHC molecules are recognized by which immune cells?
    a. CD8+ T cells
    b. CD4+ T cells
    c. B cells cells
    d. Dendritic cells
16. This class of MHC molecule presents antigens that are internalized into vesicles, and are typically derived from extracellular microbes and soluble proteins.
17. This class of MHC molecule displays peptides that are derived from proteins, such as viral and tumor antigens, that are located in the cytoplasm and usually produced within the cell.
18. What are the 4 types of hypersensitivity reactions?
19. IgE production occurs during which type of hypersensitivity?
    a. Immediate
    b. Antibody-mediated
    c. Immune complex mediated
    d. T cell mediated
20. Immediate (Type 1) hypersensitivity is associated with
    a. Arthritis
    b. Necrotizing vasculitis
    c. Anaphylaxis
    d. Tuberculin reaction
21. Bronchial smooth muscle contraction, mast cell activity, pruritus, urticaria, hypotensive shock and a second, delayed phase are all possible symptoms of which type of hypersensitivity reactions?
22. When activated, mast cells immediately release granules which can be split into all of the following categories except
    a. Vasoactive amines
    b. Antibody-antigen complexes
    c. Enzymes
    d. Proteoglycans
23. What is /what are the functions of the the most potent vasoactive amine released by mast cells (aka histamine).
24. What are the lipid mediators released/produced by mast cells and what are their functions?
25. What is the late phase reaction in relation to an immediate hypersensitivity reaction?
26. Antibodies acting against antigens present on cell surfaces or within the ECM is indicative of what type of hypersensitivity?
    a. Immediate
    b. Antibody-mediated
    c. Immune complex mediated
    d. T cell mediated
27. Provide examples of 2-3 different Type 2 (antibody-mediated) hypersensitivity diseases.

28. In myasthenia gravis, antibodies inhibit the binding of what neurotransmitter leading to muscle weakness and paralysis?
29. What disease results from antibody overstimulation of TSH receptors?
30. In what Type 2 sensitivity disease do antibodies damage both glomerular and alveolar basement membranes?
31. How does tissue damage occur in Type 3 hypersensitivity?
32. Immune-complex mediated diseases tend to be systemic but often preferentially involves what three sites?
33. Provide 2-3 examples of different Type 3 (antigen-antibody mediated) hypersensitivity diseases.
34. Systemic immune complex disease is divided into 3 phases. Place in order from first to last.
    a. Deposition of immune complexes
    b. Inflammation and tissue injury
    c. Formation of immune complexes
35. The classic morphologic manifestation of immune complex mediated hypersensitivity is what?
36. What is an Arthus reaction?
37. Type 4 hypersensitivity is the result of the action of which cells?
38. When type 4 hypersensitivity results in a delayed reaction, as seen in the Tuberculin reaction, this is due to the action of which type of cell(s)?
    a. CD4+
    b. CD8+
    c. Both of the above
    d. None of the above
39. In T cell mediated hypersensitivity where cells produce cytokines leading to inflammation, this response is caused by which type of cell(s)?
    a. CD4+
    b. CD8+
    c. Both of the above
    d. None of the above
40. The type of T cell responsible for directly killing antigen expressing target cells in Type 4 hypersensitivity reactions are
    a. CD4+
    b. CD8+
    c. Both of the above
    d. None of the above
41. What hypersensitivity response type (ie 1-4) plays a role in graft rejection?
42. Provide 2-3 examples of different Type 4 hypersensitivity diseases.
43. Define self tolerance.
44. What is central tolerance and in what tissue(s) does it take place?
45. What is anergy?
46. What autoimmune disease involves multiple organs, characterized by a vast array of autoantibodies (particularly antinuclear antibodies (ANAs)) in which injury is caused mainly by deposition of immune complexes and binding of antibodies to various cells and tissues?

47. The hallmark of Systemic Lupus Erythematosus (SLE) is
    a. The production of autoantibodies
    b. Widespread damage to small blood vessels
    c. Progressive interstitial and perivascular fibrosis in the skin and multiple organs
    d. Necrotizing inflammation of alveolar walls
48. The fundamental defect is SLE is
    a. Failure of the mechanisms that produce normal hematopoietic mechanisms
    b. Failure of the mechanisms that maintain self-tolerance
    c. Failure of the mechanisms that recognize and eliminate foreign antigens
    d. None of the above
49. Erythema along the skin of the nose and cheeks causing a characteristic 'butterfly' rash is indicative of what autoimmune disorder?
50. Sjogren syndrome results in autoimmune destruction of what tissues/organs? What are the classical symptoms of this tissue destruction?
51. Systemic sclerosis/Scleroderma is typically characterized by what three changes? What organ/tissue does it most commonly affect?
52. Systemic sclerosis is associated with vascular damage and microvascular disease with prominent effects on all of the following tissues/organs except
    a. Heart
    b. Alimentary tract
    c. Kidneys
    d. Lungs
    e. Spleen
53. What is an allograft?
54. During tissue graft recognition/rejection, the indirect pathway of allorecognition activates which class of T cells?
    a. CD4+
    b. CD8+
    c. Both of the above
    d. None of the above
55. During tissue graft recognition/rejection, the direct pathway of allorecognition activates which class of T cell?
    a. CD4+
    b. CD8+
    c. Both of the above
    d. None of the above
56. When a recipient's body has been previously sensitized to graft antigens what type of antibody mediated rejection occurs?
57. Over what timeframe do hyperacute, acute rejection occur?
58. What causes graft-versus-host-disease (GVHD) to occur?
59. Differentiate between primary and secondary immunodeficiency syndromes.
60. Defects in leukocyte adhesion and/or the complement system are indicative of _____ (primary/secondary) immunodeficiency.
61. Severe combined immunodeficiency (SCID) represents a constellation of genetically distinct syndromes, all having in common defects in

    a. Humoral immune responses
    b. Cell-mediated immune responses
    c. Antibody-mediated immune responses
    d. Both humoral and cell-mediated immune responses
62. What is x-linked agammaglobulinemia/Bruton Agammaglobulinemia characterized by?
63. Why does x-linked agammaglobulinemia/Bruton Agammaglobulinemia typically not become apparent until about 6 months of age?
64. What is DiGeorge syndrome characterized by?
65. A lack of opsonizing IgG antibodies (as well as overall deficiency in IgA and IgE) leading to recurrent bacterial infections is common in what disorder?
    a. Hyper IgM syndrome
    b. Isolated IgA deficiency
    c. Common variable immunodeficiency
    d. X-linked agammaglobulinemia (Bruton's agammaglobulinemia)
66. What occurs in Isolated IgA deficiency? What are 3 areas infections tend to occur because of this?
67. What is Wiskott-Aldrich syndrome and Ataxia Telangiectasia?
68. What is the most common secondary immunodeficiency?
69. What are the three major routes of transmission of HIV?
70. Which type of immune cells are targeted in HIV infected individuals?
71. B cell dysfunction is seen in HIV infected patients. True or false?
72. Other than the immune/lymphoid system, what other system is a major target of HIV infection?
73. What occurs at each of the following points in the clinical course of HIV infection: acute (early), chronic infection and AIDS?
74. Dramatic increases in plasma virus, presence of serious opportunistic infections, secondary neoplasms or clinical neurologic disease occurs during which stage of HIV infection?
    a. AIDS
    b. Chronic infection
    c. Acute infection
    d. All of the above
75. What accounts (generally speaking) for the majority of deaths in untreated AIDS patients?
76. What is the most common fungal infection in AIDS patients?
77. What are the most common tumors (4) that develop in AIDS patients and what is a common feature that they all share?
78. What are some of the common neurologic infections seen in AIDS patients?
79. Most common neoplasm of AIDS patients is?
    a. B cell non-Hodgkin lymphoma
    b. Cervical cancer
    c. Kaposi sarcoma
    d. Anal cancer
80. Extracellular deposits of fibrillar proteins responsible for pressure atrophy related tissue damage and functional compromise describes what condition?

81. How is amyloid most commonly identified histologically?
82. What are the 3 most common forms of amyloid, where do they come from and where are they found?
83. Amyloidosis results from
    a. Excessive cell-mediated immune activity resulting in proliferation of active Ig secreting CD8+ cell populations
    b. Abnormal folding of starches, which become insoluble, aggregate, and deposit as fibrils in extracellular tissues
    c. Abnormal folding of proteins, which become insoluble, aggregate, and deposit as fibrils in extracellular tissues
    d. None of the above
84. What is the most common subtype of amyloidosis?
85. Differentiate between primary and secondary amyloidosis.
86. In what condition do you expect to find Bence-Jones protein? Where are they typically found?
87. Although amyloidosis associated with plasma cell proliferations cannot reliably be distinguished from the secondary form by its organ distribution, it more often involves which tissues?
88. What is the gross appearance of an organ which has accumulated a large amount of amyloid?
89. Where is amyloid seen histologically?

1. D (pg 186-187)
2. Innate immunity (pg 187)
3. Adaptive (pg 186, 188)
4. Pattern recognition receptors are located in all cellular compartments where microbes may be present. These receptors recognize certain microbial components that are shared between related microbes and are often responsible for infectivity (and thus cannot be mutated to all microbes to escape defense mechanisms). Toll-like receptors are one the best known classes of pattern recognition receptors and are present on cell membranes and endosomes. Ultimately these receptors are responsible for stimulating recruitment and activation of leukocytes (pg 187)
5. C (pg 189)
6. Humoral (pg 189)
7. B cells/plasma cells (pg 191)
8. D (pg 191)
9. A (pg 192)
10. B (pg 193)
11. C (pg 193)
12. B (pg 193-194)
13. MHC molecules display peptide fragments of protein antigens for recognition by antigen-specific T cells. In humans these are referred to as human leukocyte antigens (pg 194)
14. All nucleated cells and platelets (pg 194)
15. A (pg 194)
16. Class 2 (pg 194)
17. Class 1 (pg 194)
18. Type 1 - immediate; type 2 - antibody-mediated; type 3 - immune complex mediated; type 4 - t cell mediated (pg 200-201)
19. A (pg 201/202)
20. C (pg 201/202)
21. Immediate hypersensitivity/Type 1 (pg 201)
22. B (pg 203)
23. Increased vascular permeability; intense smooth muscle contraction; increased secretions from nasal, bronchial and gastric glands (ptg 203)
24. Leukotrienes (most potent vasoactive and spasmogenic agents known - several thousand times more active than histamine in increasing vascular permeability and causing bronchial smooth muscle contraction); prostaglandin D2 (causes bronchospasm and increased mucus secretion); and platelet-activating factor (PAF, causes platelet aggregation, release of histamine, bronchospasm, increased vascular permeability and vasodilation) (pg 203)
25. Leukocytes are recruited that amplify and sustain the inflammatory response without additional exposure to the triggering antigen (pg 204)
26. B (pg 206)
27. Autoimmune hemolytic anemia, autoimmune thrombocytopenic purpura, pemphigus vulgaris, vasculitis caused by ANCA, goodpasture syndrome, acute rheumatic fever, myasthenia gravis, graves disease, insulin-resistant diabetes, pernicious anemia (pg 206)

28. Acetylcholine (pg 206)
29. Graves disease (pg 206)
30. Goodpasture syndrome (pg 206)
31. Antigen-antibody complexes produce tissue damage mainly by eliciting inflammation at the sites of deposition (pg 207)
32. Kidneys, joints and small blood vessels (pg 207)
33. Systemic lupus erythematosus, poststreptococcal glomerulonephritis, polyarteritis nodosa, reactive arthritis, serum sickness, arthus reaction (pg 207)
34. C, A, B (pg 207-208)
35. Acute vasculitis associated with necrosis of the vessel wall and intense neutrophilic infiltration. Necrotic tissue and deposits of immune complexes, complement, and plasma protein appear as a smudgy eosinophilic area of tissue destruction, an appearance termed fibrinoid necrosis (pg 208)
36. Localized area of tissue necrosis resulting from acute immune complex vasculitis, usually elicited in the skin (pg 208)
37. Inflammation resulting from cytokines produced by CD4+ T cells and cell killing by CD8+ T cells (pg 208)
38. A (pg 209-210)
39. A (pg 209)
40. B (pg 211)
41. T cell mediated (Type 4) hypersensitivity (pg 211)
42. Type 1 diabetes mellitus, multiple sclerosis, tuberculosis, inflammatory bowel disease, poison ivy, psoriasis (pg 209)
43. Self tolerance is the non reaction of immune cells against host/self antigens. It is what allows immune cells to live in harmony with the rest of the body's tissues (pg 212)
44. Central tolerance is the elimination of immature self reactive T and B lymphocytes and it takes place in the thymus and bone marrow. Some cells are eliminated, some are rendered harmless and some switch to new antigen receptors that are not self-reactive (pg 212-213)
45. Functional inactivation of lymphocytes that recognize self antigens (pg 213)
46. Systemic Lupus Erythematosus (pg 218)
47. A (pg 219)
48. B (pg 219)
49. Lupus (pg 224)
50. Salivary and lacrimal gland destruction. Xerostomia (dry mouth) and keratoconjunctivitis sicca (dry eyes) (pg 226)
51. (1) chronic inflammation thought to be a result of autoimmunity, (2) widespread damage to small blood vessels and (3) progressive interstitial and perivascular fibrosis of the skin and multiple organs. Most commonly affects the skin (pg 228)
52. E (pg 229)
53. Grafts exchanged between individuals of the same species (pg 231)
54. A (pg 232)
55. C (pg 232)
56. Hyperacute rejection (pg 233)

57. Hyperacute - minutes to hours; acute - within days (in an untreated recipient) to suddenly months or even years later after immunosuppression is tapered/terminated (233-234)
58. GVHD occurs when when immunologically competent cells or their precursors are transplanted into immunologically crippled recipients, and the transferred cells recognize alloantigens in the host and attack host tissues → donated immune cells are transplanted into immunocompromised patient and start attacking the “new” host tissues (pg 236)
59. Primary are congenital syndromes which are genetically determined whereas secondary are acquired and may arise as a complication of cancers, infections, malnutrition or side effects of immunosuppression, irradiation or chemotherapy (pg 237)
60. Primary (pg 239)
61. D (pg 239)
62. Failure of B-cell precursors) to develop into mature B cells (pg 240)
63. By ~6 months, maternal immunoglobulins are depleted (pg 240)
64. It is a 22q11 deletion characterized primarily by T cell deficiency resulting from failure of development of the thymus. Other symptoms include tetany (absence of the parathyroids) as well as congenital defects of the heart and great vessels (among other tissues (pg 241)
65. A (pg 241)
66. Low levels of both serum and secretory IgA result in infections occurring in the respiratory, gastrointestinal and urogenital tracts. This is because IgA is the major antibody in external secretions(pg 242)
67. In Wiskott-Aldrich, there is thrombocytopenia, eczema and predisposition to recurrent infection leading to death. Early on the thymus is morphologically normal but peripheral and lymph node T cell levels progressively decrease. Prone to B cell lymphomas. In ataxia telangiectasia, there is abnormal gait (ataxia), vascular malformations (telangiectasia), neurologic deficits, increased incidence of tumors and immunodeficiency which may affect both B and T cells. Defective antibody production and thymic hypoplasia are often seen.
68. AIDS (pg 243)
69. Sexual contact, parenteral inoculation and passage of the virus from infected mothers to their newborns (pg 244)
70. CD4+ helper T cells are the primary targets of the infection. Although macrophages and dendritic cells are also targets of HIV infection (pg 245)
71. True (pg 249)
72. Central nervous system (pg 245, 250)
73. Acute - infection of memory CD4+ T cells and death of many infected cells. Mucosal infection is followed by dissemination of the virus and the development of host immune response. HIV particles are present in the blood. Chronic - lymph nodes and the spleen are sites of continuous HIV replication and cell destruction. Few/no clinical manifestations of the HIV infections are present (clinical latency period). Most peripheral CD4+ cells do not contain the virus but there is continuous destruction of CD4+ T cells within lymphoid tissue, leading to slow decline of CD4+ numbers, despite continuous creation of new T cells. As T cell losses accumulate, host

defenses begin to wane and more and more surviving T cells contain HIV. AIDS - breakdown of host defense, dramatic increase in plasma virus and severe, life-threatening clinical disease. Opportunistic infections, secondary neoplasms or clinical neurologic disease will occur (pg 250-252)

74. A (pg 250-252)
75. Opportunistic infections (pg 252)
76. Candidiasis (pg 253)
77. Kaposi sarcoma, B cell lymphoma, cervical cancer and anal cancer in men. These cancers are all caused by oncogenic DNA viruses - Kaposi sarcoma herpesvirus/human herpesvirus 8 (Kaposi sarcoma), EBV (B-cell lymphoma), and human papillomavirus (cervical and anal carcinoma (pg 253)
78. Cryptococcosis, Toxoplasma gondii, JC virus (pg 253)
79. C (pg 253)
80. Amyloidosis (pg 256)
81. Apple green birefringence under polarized light with a congo red stain (pg 257)
82. 1) Amyloid light chain (AL) which is made up of complete immunoglobulins, fragments of light chains (aka part of immunoglobulins) or both; 2) amyloid-associated (AA) protein derived from a non-Ig protein made by the liver which is typically seen in states of chronic inflammation, also called secondary amyloidosis; 3) beta-amyloid protein (AB) which constitutes the core of cerebral plaques found in alzheimer disease as well as amyloid deposited in walls of cerebral blood vessels in individuals with this disease (pg 257)
83. C (pg 258)
84. Amyloid light chain (AL) (pg 258)
85. Primary - when amyloidosis is associated with some plasma cell disorder. Secondary - occurs as a complication of an underlying chronic inflammatory or tissue-destructive process (pg 258)
86. AL type amyloidosis, commonly secondary to multiple myeloma. Found excreted in the urine but can also be detected in serum (pg 258)
87. Heart, GI tract, respiratory tract, peripheral nerves, skin and tongue (pg 260)
88. Enlarged, with a grey, waxy and firm consistency (pg 260)
89. Deposition is always extracellular and begins between cells (pg 260)