

龔瑞林老師的考試題目提要

Terminology for Examination

Acquired immunity
addressins
adjuvants
Alpha 2-macroglobulin
Affinity maturation
Allelic exclusion
allotypes
anergy
Antigen presentation
antigen-presenting cells
Antigen processing
AP-1
autocrine/paracrine
autoimmunity
B7
beta2-microglobulin
C3/C5 convertase
Catalytic antibody
CD28
CD4/CD8
Cell mediated immunity
class II-associated invariant chain peptide (CLIP)
Class switching
Clonal deletion
<u>clonal election theory</u>
complementarity-determining regions
Complement factor P/H/I/DAF
Complement fixation/activation
co-receptors
co-stimulators
C-reactive protein
Dendritic cells
Disseminated intravascular coagulation
Double negative/positive thymocytes
endocytosis/phagocytosis

epitope
Fab/Fcregion
GALT/MALT
Coblet cell
granulocytes
granzyme/perforin
heavy/lightchain
helper/cytotoxic/suppressor T cells
hematopoisis
HLA-DM
homing
humoral/cellular immunity
hybridoma
Idiotype anti-idiotypic network
inflammation
Inflammatory cytokines/IL-6/IL-12/TNF-alpha/chemokine
Immunoglobulin superfamily
innate/adaptive immunity
integrins
interleukins
isotypes
ITAMs
Junctional diversity
LAF-1/ICAMs
Langerhans' cell
Lck/Fyn
linear/conformational/discontinuous epitopes
Major histocompatibility complex
Mast cell
megakaryocytes
Membrane attack complex
MHC classI/II
MHC restriction
Mixed lymphocyte reaction
Monoclonal antibody
Natural killer cells
necrosis/apoptosis

NFAT
NF kappaB
Natur killer cells
Opportunistic pathogen
opsonin
opsonization
Paneth cell
passive/active immunity
Periarteriolar lymphoid sheath
Peyer's patches
phagolysosome
phagocytosis
phagolysosome
Pluripotent hematopoietic stem cell
P nucleotides
postive/negative selection
<u>primary/secondary lymphoid tissue</u>
pro/pre/immature/mature/naïve/virgin/plasma/memoryBcell
Professional antigen-presenting cell
proteasome
selectin
Sepsis/septic shock
Somatic hypermutation
STATs
suppressor/regulatory CD4 T cells
Surrogate light chain
Terminal deoxynucleotidyl transferase
Th1/Th2
Thymic selection
thymocytes
Tissue typing
Toll-like receptor
translocation/proto-oncogenes
Transporter associated with antigen processing(TAP)
variable/constant regions
ZAP-70

Chapter 3			
Principles of Adaptive Immunity		71	
3-1	Innate and adaptive immunity differ in their strategies for pathogen recognition	71	
3-2	Immunoglobulins and T-cell receptors are the highly variable recognition molecules of adaptive immunity	72	
3-3	The diversity of immunoglobulins and T-cell receptors is generated by gene rearrangement	73	
3-4	Clonal selection of B and T lymphocytes is the guiding principle of the adaptive immune response	75	
3-5	Adaptive immune responses are initiated in secondary lymphoid tissues by antigen-bearing dendritic cells and T cells	75	
3-6	T-cell receptors recognize degraded fragments of pathogen proteins	76	
3-7	T-cell receptors recognize peptide antigens bound to human cell-surface molecules	78	
3-8	Two classes of MHC molecule present peptide antigens to two types of T cell	78	
3-9	MHC class I molecules present antigens of intracellular origin to CD8 T cells	80	
3-10	MHC class II molecules present antigens of extracellular origin to CD4 T cells	81	
3-11	Effector CD4 T cells help B cells become antibody-producing plasma cells	81	
3-12	Extracellular pathogens and their toxins are eliminated by antibodies	83	
3-13	Antibody quality improves during the course of an adaptive immune response	85	
3-14	Immunological memory is a consequence of clonal selection	86	
3-15	Clonal selection makes T cells and B cells tolerant of self and responsive to pathogens	87	
3-16	Unwanted effects of adaptive immunity cause autoimmune disease, transplant rejection and allergy	88	
	Summary to Chapter 3	90	
	Questions	91	
Chapter 4			
Antibody Structure and the Generation of B-Cell Diversity		95	
	The structural basis of antibody diversity	96	
4-1	Antibodies are composed of polypeptides with variable and constant regions	96	
4-2	Immunoglobulin chains are folded into compact and stable protein domains	97	
4-3	An antigen-binding site is formed from the hypervariable regions of a heavy-chain V domain and a light-chain V domain	99	
4-4	Antigen-binding sites vary in shape and physical properties	100	
4-5	Monoclonal antibodies are produced from a clone of antibody-producing cells	102	
4-6	Monoclonal antibodies are used as treatments for a variety of diseases	104	
	Summary	105	
	Generation of immunoglobulin diversity in B cells before encounter with antigen	105	
4-7	The DNA sequence encoding a V region is assembled from two or three gene segments	106	
4-8	Random recombination of gene segments produces diversity in the antigen-binding sites of immunoglobulins	106	
4-9	Recombination enzymes produce additional diversity in the antigen-binding site	108	
4-10	Developing and naive B cells use alternative mRNA splicing to make both IgM and IgD	110	
4-11	Each B cell produces immunoglobulin of a single antigen specificity	111	
4-12	Immunoglobulin is first made in a membrane-bound form that is present on the B-cell surface	111	
	Summary	112	
	Diversification of antibodies after B cells encounter antigen	113	
4-13	Secreted antibodies are produced by an alternative pattern of heavy-chain RNA processing	113	
4-14	Rearranged V-region sequences are further diversified by somatic hypermutation	114	
4-15	Isotype switching produces immunoglobulins with different C regions but identical antigen specificities	115	
4-16	Antibodies with different C regions have different effector functions	117	
	Summary	119	
	Summary to Chapter 4	119	
	Questions	121	
Chapter 5			
Antigen Recognition by T Lymphocytes		125	
	T-cell receptor diversity	126	
5-1	The T-cell receptor resembles a membrane-associated Fab fragment of immunoglobulin	126	
5-2	T-cell receptor diversity is generated by gene rearrangement	127	
5-3	The <i>RAG</i> genes were key elements in the origin of adaptive immunity	129	
5-4	Expression of the T-cell receptor on the cell surface requires association with additional proteins	129	
5-5	A distinct population of T cells expresses a second class of T-cell receptor with γ and δ chains	130	
	Summary	131	
	Antigen processing and presentation	132	
5-6	The two classes of MHC molecule present antigen to CD8 and CD4 T cells, respectively	133	
5-7	The two classes of MHC molecule have similar three-dimensional structures	134	
5-8	MHC molecules bind a variety of peptides	135	
5-9	MHC class I and class II molecules bind peptides in different intracellular compartments	137	
5-10	Peptides generated in the cytosol are transported into the endoplasmic reticulum, where they bind MHC class I molecules	137	

5-11	MHC class I molecules bind antigenic peptide as part of a peptide-loading complex	138		
5-12	Peptides presented by MHC class II molecules are generated in acidified intracellular vesicles	140		
5-13	MHC class II molecules are prevented from binding peptides in the endoplasmic reticulum by the invariant chain	140		
5-14	The T-cell receptor specifically recognizes both peptide and MHC molecule	142		
5-15	The two classes of MHC molecule are expressed differentially on cells	143		
5-16	Cross-presentation allows extracellular antigens to be presented by MHC class I	144		
	Summary	145		
	The major histocompatibility complex	145		
5-17	The diversity of MHC molecules in the human population is due to multigene families and genetic polymorphism	146		
5-18	The HLA class I and class II genes occupy different regions of the HLA complex	147		
5-19	Other proteins involved in antigen processing and presentation are encoded in the HLA class II region	148		
5-20	MHC polymorphism affects the binding and presentation of peptide antigens to T cells	149		
5-21	MHC diversity results from selection by infectious disease	151		
5-22	MHC polymorphism triggers T-cell reactions that can reject transplanted organs	153		
	Summary	154		
	Summary to Chapter 5	154		
	Questions	155		
Chapter 6				
	The Development of B Lymphocytes	159		
	The development of B cells in the bone marrow	160		
6-1	B-cell development in the bone marrow proceeds through several stages	160		
6-2	B-cell development is stimulated by bone marrow stromal cells	161		
6-3	Pro-B cell rearrangement of the heavy-chain locus is an inefficient process	162		
6-4	The pre-B-cell receptor monitors the quality of immunoglobulin heavy chains	163		
6-5	The pre-B-cell receptor causes allelic exclusion at the immunoglobulin heavy-chain locus	164		
6-6	Rearrangement of the light-chain loci by pre-B cells is relatively efficient	165		
6-7	B cells have to pass two main checkpoints in their development in the bone marrow	167		
6-8	A program of protein expression underlies the stages of B-cell development	168		
6-9	Many B-cell tumors carry chromosomal translocations that join immunoglobulin genes to genes that regulate cell growth	171		
6-10	B cells expressing the glycoprotein CD5 express a distinctive repertoire of receptors	171		
	Summary	173		
	Selection and further development of the B-cell repertoire	174		
6-11	The population of immature B cells is purged of cells bearing self-reactive B-cell receptors	174		
6-12	The antigen receptors of autoreactive immature B cells can be modified by receptor editing	175		
6-13	Immature B cells specific for monovalent self antigens are made nonresponsive to antigen	176		
6-14	Maturation and survival of B cells requires access to lymphoid follicles	177		
6-15	Encounter with antigen leads to the differentiation of activated B cells into plasma cells and memory B cells	179		
6-16	Different types of B-cell tumor reflect B cells at different stages of development	180		
	Summary	182		
	Summary to Chapter 6	182		
	Questions	184		
Chapter 7				
	The Development of T Lymphocytes	187		
	The development of T cells in the thymus	187		
7-1	T cells develop in the thymus	188		
7-2	Thymocytes commit to the T-cell lineage before rearranging their T-cell receptor genes	190		
7-3	The two lineages of T cells arise from a common thymocyte progenitor	191		
7-4	Gene rearrangement in double-negative thymocytes leads to assembly of either a $\gamma\delta$ receptor or a pre-T-cell receptor	193		
7-5	Thymocytes can make four attempts to rearrange a β -chain gene	194		
7-6	Rearrangement of the α -chain gene occurs only in pre-T cells	195		
7-7	Stages in T-cell development are marked by changes in gene expression	196		
	Summary	197		
	Positive and negative selection of the T-cell repertoire	198		
7-8	T cells that recognize self-MHC molecules are positively selected in the thymus	199		
7-9	Continuing α -chain gene rearrangement increases the chance for positive selection	200		
7-10	Positive selection determines expression of either the CD4 or the CD8 co-receptor	200		
7-11	T cells specific for self antigens are removed in the thymus by negative selection	202		
7-12	Tissue-specific proteins are expressed in the thymus and participate in negative selection	202		
7-13	Regulatory CD4 T cells comprise a distinct lineage of CD4 T cells	203		
7-14	T cells undergo further differentiation in secondary lymphoid tissues after encounter with antigen	203		
7-15	Most T-cell tumors represent early or late stages of T-cell development	204		
	Summary	205		
	Summary to Chapter 7	205		
	Questions	207		

Chapter 8**T Cell-Mediated Immunity****211**

	Activation of naive T cells on encounter with antigen	212
8-1	Dendritic cells carry antigens from sites of infection to secondary lymphoid tissues	212
8-2	Dendritic cells are adept and versatile at processing antigens from pathogens	213
8-3	Naive T cells first encounter antigen presented by dendritic cells in secondary lymphoid tissues	215
8-4	Homing of naive T cells to secondary lymphoid tissues is determined by chemokines and cell-adhesion molecules	216
8-5	Activation of naive T cells requires a co-stimulatory signal delivered by a professional antigen-presenting cell	219
8-6	Secondary lymphoid tissues contain three kinds of professional antigen-presenting cell	220
8-7	When T cells are activated by antigen, signals from T-cell receptors and co-receptors alter the pattern of gene transcription	222
8-8	Proliferation and differentiation of activated T cells are driven by the cytokine interleukin-2	224
8-9	Antigen recognition by a naive T cell in the absence of co-stimulation leads to the T cell becoming nonresponsive	226
8-10	On activation, CD4 T cells acquire distinctive helper functions	227
8-11	Naive CD8 T cells are activated to become cytotoxic effector cells in several different ways	229
	Summary	230
	The properties and functions of effector T cells	231
8-12	Effector T-cell responses to infection do not depend on co-stimulatory signals	231
8-13	Effector T-cell functions are carried out by cytokines and cytotoxins	232
8-14	Cytotoxic CD8 T cells are selective and serial killers of target cells at sites of infection	234
8-15	Cytotoxic T cells kill their target cells by inducing apoptosis	236
8-16	T _H 1 CD4 cells induce macrophages to become activated	237
8-17	T _H 1 cells coordinate the host response to pathogens that live in macrophages	239
8-18	CD4 T _H 2 cells activate only those B cells that recognize the same antigen as they do	241
8-19	Regulatory CD4 T cells limit the activities of effector CD4 and CD8 T cells	242
	Summary	243
	Summary to Chapter 8	245
	Questions	245

Chapter 9**Immunity Mediated by B Cells and Antibodies****249**

	Antibody production by B lymphocytes	250
9-1	B-cell activation requires cross-linking of surface immunoglobulin	250

9-2	B-cell activation requires signals from the B-cell co-receptor	250
9-3	The antibody response to certain antigens does not require T-cell help	251
9-4	Activation of naive B cells by most antigens requires help from CD4 T cells	254
9-5	The primary focus of clonal expansion in the medullary cords produces plasma cells secreting IgM	256
9-6	Follicular dendritic cells provide long-lasting depositories of B-cell antigens	256
9-7	Activated B cells undergo somatic hypermutation and isotype switching in the specialized microenvironment of the B-cell zone	257
9-8	Selection of centrocytes by antigen in the germinal center drives affinity maturation of the B-cell response	259
9-9	The cytokines made by helper T cells determine how B cells switch their immunoglobulin isotype	261
9-10	Cytokines made by helper T cells determine the differentiation of antigen-activated B cells into plasma cells or memory cells	262
	Summary	262
	Antibody effector functions	263
9-11	IgM, IgG, and monomeric IgA protect the internal tissues of the body	264
9-12	Dimeric IgA protects the mucosal surfaces of the body	266
9-13	IgE provides a mechanism for the rapid ejection of pathogens from the body	267
9-14	Mothers provide protective antibodies to their young, both before and after birth	268
9-15	High-affinity neutralizing antibodies prevent viruses and bacteria from infecting cells	268
9-16	High-affinity IgG and IgA antibodies are used to neutralize microbial toxins and animal venoms	270
9-17	Binding of IgM to antigen on a pathogen's surface activates complement by the classical pathway	272
9-18	Two forms of C4 tend to be fixed at different sites on pathogen surfaces	273
9-19	Complement activation by IgG requires the participation of two or more IgG molecules	274
9-20	Erythrocytes facilitate the removal of immune complexes from the circulation	275
9-21	The four subclasses of IgG have different and complementary functions	275
9-22	Fc receptors enable hematopoietic cells to bind and be activated by IgG bound to pathogens	278
9-23	A variety of low-affinity Fc receptors are specific for IgG	280
9-24	IgE binds to high-affinity Fc receptors on mast cells, basophils, and activated eosinophils	282
9-25	The Fc receptor for monomeric IgA belongs to a different family from the Fc receptors for IgG and IgE	284
	Summary	285
	Summary to Chapter 9	285
	Questions	286

Chapter 10**The Body's Defenses Against Infection 289**

Preventing infection at mucosal surfaces 290	
10-1 The communication functions of mucosal surfaces render them vulnerable to infection	290
10-2 The gastrointestinal tract is invested with distinctive secondary lymphoid tissues	291
10-3 M cells and dendritic cells facilitate transport of microbes from the gut lumen to gut-associated lymphoid tissues	293
10-4 Effector lymphocytes populate healthy mucosal tissue in the absence of infection	294
10-5 B cells and T cells commit to mucosal lymphoid tissues after they encounter their specific antigen	296
10-6 Effector lymphocytes activated in any one mucosal tissue recirculate through all mucosal tissues	298
10-7 Dimeric IgA binds pathogens at various sites in mucosal tissues	298
10-8 Two subclasses of IgA have complementary properties for controlling microbial populations	299
10-9 Humans with selective deficiency of IgA do not succumb to infection	300
10-10 Intestinal epithelial cells contribute to innate defense of the gut	301
10-11 Intestinal helminth infections provoke strong T_H2 -mediated immune responses	301
Summary	302
Immunological memory and the secondary immune response 303	
10-12 The antibodies formed during a primary immune response prevent reinfection for several months after disease	304
10-13 Immunological memory is sustained by clones of long-lived memory T cells and B cells	305
10-14 Vaccination against a pathogen can generate immunological memory that persists for life	306
10-15 Pathogen-specific memory B cells are more abundant and make better antibodies than do naive B cells	307
10-16 Activation of a secondary response involves cell–cell interactions like those activating the primary response	308
10-17 Only memory B cells, and not naive B cells, participate in the secondary immune response	308
10-18 Immune-complex mediated inhibition of naive B cells is used to prevent hemolytic anemia of the newborn	309
10-19 In the response to influenza virus, immunological memory is gradually eroded	310
10-20 Several cell-surface markers distinguish memory T cells from naive T cells	311
10-21 Two types of memory T cell function in different tissues	313
10-22 Maintenance of immunological memory is not dependent on antigen	313
Summary	314

Bridging innate and adaptive immunity 315

10-23 $\gamma\delta$ T cells contribute to the innate immune response	315
10-24 Individual NK cells express many different combinations of receptors belonging to one of two receptor families	317
10-25 NK cells use receptors for MHC class I molecules to identify infected cells	318
10-26 NK cells have inhibitory receptors with different specificities for MHC class I molecules	319
10-27 Inhibitory receptors for self MHC class I make NK cells tolerant to self and responsive to loss of MHC class I	321
10-28 T cells recognizing lipid antigens protect against mycobacterial infection	322
10-29 NK T cells are cells of innate immunity that express $\alpha\beta$ T-cell receptors	323
Summary	323
Summary to Chapter 10 324	
Questions 326	

Chapter 11**Failures of the Body's Defenses 329**

Evasion and subversion of the immune system by pathogens 329	
11-1 Genetic variation within some species of pathogens prevents effective long-term immunity	330
11-2 Mutation and recombination allow influenza virus to escape from immunity	330
11-3 Trypanosomes use gene rearrangement to change their surface antigens	332
11-4 Herpesviruses persist in human hosts by hiding from the immune response	333
11-5 Certain pathogens sabotage or subvert immune defense mechanisms	334
11-6 Bacterial superantigens stimulate a massive but ineffective T-cell response	336
11-7 Immune responses can contribute to disease	337
Summary	337
Inherited immunodeficiency diseases 338	
11-8 Rare primary immunodeficiency diseases reveal how the human immune system works	338
11-9 Inherited immunodeficiency diseases are caused by dominant, recessive, or X-linked gene defects	339
11-10 Recessive and dominant mutations in the interferon- γ receptor cause diseases of differing severity	340
11-11 Antibody deficiency leads to an inability to clear extracellular bacteria	341
11-12 Diminished production of antibodies also results from inherited defects in T-cell help	343
11-13 Defects in complement components impair antibody responses and cause the accumulation of immune complexes	343
11-14 Defects in phagocytes result in enhanced susceptibility to bacterial infection	345
11-15 Defects in T-cell function result in severe combined immune deficiencies	346

11-16	Some inherited immunodeficiencies lead to specific disease susceptibilities	348	12-13	Urticaria, angioedema, and eczema are allergic reactions in the skin	381
11-17	Transplantation of hematopoietic stem cells is used to correct genetic defects of the immune system	349	12-14	Food allergies cause systemic effects as well as gut reactions	382
	Summary	351	12-15	People with parasite infections and high levels of IgE rarely develop allergic disease	383
	Acquired immune deficiency syndrome	351	12-16	Allergic reactions are prevented and treated by three complementary approaches	384
11-18	HIV is a retrovirus that causes slowly progressing disease	351		Summary	385
11-19	HIV infects CD4 T cells, macrophages, and dendritic cells	352		Type II, III, and IV hypersensitivity reactions	386
11-20	Most people who become infected with HIV progress in time to develop AIDS	353	12-17	Type II hypersensitivity reactions are caused by antibodies specific for altered components of human cells	386
11-21	Genetic deficiency of the CCR5 co-receptor for HIV confers resistance to infection	356	12-18	To avoid type II hypersensitivity reactions in blood transfusion, donors and recipients are matched for ABO antigens	387
11-22	HLA and KIR polymorphisms influence the progression to AIDS	356	12-19	Type III hypersensitivity reactions are caused by immune complexes formed from IgG and soluble antigens	389
11-23	HIV escapes the immune response and develops resistance to antiviral drugs by rapid mutation	357	12-20	Systemic disease caused by immune complexes can follow the administration of large quantities of soluble antigens	390
11-24	Clinical latency is a period of active infection and renewal of CD4 T cells	358	12-21	Inhaled antigens can cause type III hypersensitivity reactions	392
11-25	HIV infection leads to immunodeficiency and death from opportunistic infections	359	12-22	Type IV hypersensitivity reactions are mediated by antigen-specific effector T cells	392
	Summary	360	12-23	Celiac disease is caused by hypersensitivity to common food proteins	395
	Summary to Chapter 11	361	12-24	Severe hypersensitivity reactions to certain drugs are strongly correlated with HLA class I allotypes	397
	Questions	361		Summary	398
	Chapter 12			Summary to Chapter 12	398
	Over-reactions of the Immune System	365		Questions	399
12-1	Four types of hypersensitivity reaction are caused by different effector mechanisms of adaptive immunity	365		Chapter 13	
	Type I hypersensitivity reactions	367		Disruption of Healthy Tissue by the Immune Response	403
12-2	IgE binding to FcεR1 provides mast cells, basophils, and activated eosinophils with antigen receptors	367		Autoimmune diseases	403
12-3	Mast cells defend and maintain the tissues where they live	368	13-1	In healthy individuals the immune system is tolerant of self antigens	404
12-4	Tissue mast cells orchestrate IgE-mediated allergic reactions through the release of inflammatory mediators	370	13-2	Autoimmune diseases are caused by the loss of tolerance to self antigens	404
12-5	Eosinophils are specialized granulocytes that release toxic mediators in IgE-mediated responses	371	13-3	The effector mechanisms of autoimmunity resemble those causing hypersensitivity reactions	405
12-6	Basophils are rare granulocytes that initiate T _H 2 responses and the production of IgE	373	13-4	Endocrine glands contain specialized cells that are targets for organ-specific autoimmunity	407
12-7	Very few antigens that enter the human body are allergens that stimulate an IgE response	374	13-5	Autoimmune diseases of the thyroid can cause either underproduction or overproduction of thyroid hormones	408
12-8	Predisposition to allergic disease has a genetic basis	376	13-6	Ectopic lymphoid tissue can form at sites inflamed by autoimmune disease	409
12-9	IgE-mediated allergic reactions consist of an immediate response followed by a late-phase response	377	13-7	The cause of an autoimmune disease can be revealed by the transfer of the disease by immune effectors	410
12-10	The effects of IgE-mediated allergic reactions vary with the site of mast-cell activation	378	13-8	Type 1 diabetes is caused by the selective destruction of insulin-producing cells in the pancreas	411
12-11	Systemic anaphylaxis is caused by allergens in the blood	379			
12-12	Rhinitis and asthma are caused by inhaled allergens	380			

13-9	Autoantibodies against common components of human cells can cause systemic autoimmune disease	412	14-5	The need for a vaccine and the demands placed on it change with the prevalence of the disease	444
13-10	Most rheumatological diseases are caused by autoimmunity	413	14-6	Vaccines have yet to be found for many chronic pathogens	446
13-11	Rheumatoid arthritis can be treated with monoclonal antibodies that target either TNF- α or B cells	413	14-7	Genome sequences of human pathogens open up new avenues of vaccine design	447
13-12	Multiple sclerosis and myasthenia gravis are autoimmune diseases of the nervous system	414	14-8	A useful vaccine against HIV has yet to be found	449
	Summary	416	14-9	An effective and acceptable rotavirus vaccine has been developed	450
	Genetic and environmental factors predispose to autoimmune disease	417	14-10	Vaccine development faces greater public scrutiny than drug development	450
13-13	All autoimmune diseases involve breaking T-cell tolerance	417		Summary to Chapter 14	451
13-14	Incomplete deletion of self-reactive T cells in the thymus causes autoimmune disease	417		Questions	452
13-15	Insufficient control of T-cell co-stimulation favors autoimmunity	418		Chapter 15	
13-16	Regulatory T cells protect cells and tissues from autoimmunity	419		Transplantation of Tissues and Organs	455
13-17	HLA is the dominant genetic factor affecting susceptibility to autoimmune disease	420	15-1	Transplant rejection and graft-versus-host reaction are immune responses caused by genetic differences between transplant donors and recipients	455
13-18	Different combinations of HLA class II allotypes confer susceptibility and resistance to diabetes	422	15-2	Blood transfusion is the most widespread kind of transplantation in clinical medicine	456
13-19	Autoimmunity is initiated by disease-associated HLA allotypes presenting antigens to autoimmune T cells	424		Transplantation of solid organs	458
13-20	Noninfectious environmental factors influence the course of autoimmune diseases	425	15-3	Antibodies against ABO or HLA antigens cause hyperacute rejection of transplanted organs	458
13-21	Genetic and environmental effects combine to cause one form of rheumatoid arthritis	426	15-4	Anti-HLA antibodies can arise from pregnancy, blood transfusion, or previous transplants	459
13-22	Infections are environmental factors that can trigger autoimmune disease	427	15-5	Organ transplantation involves procedures that inflame the donated organ and the transplant recipient	460
13-23	Autoimmune T cells can be activated in a pathogen-specific or nonspecific manner by infection	428	15-6	Acute rejection is caused by effector T cells responding to HLA differences between donor and recipient	460
13-24	In the course of autoimmune disease the specificity of the autoimmune response broadens	430	15-7	HLA differences between transplant donor and recipient activate numerous alloreactive T cells	462
13-25	Senescence of the T-cell population can contribute to autoimmunity	432	15-8	Negative selection in the thymus limits the number of expressed MHC isoforms	462
13-26	Does the current increase in hypersensitivity and autoimmune disease have a common cause?	432	15-9	Chronic rejection of organ transplants is due to the indirect pathway of allorecognition	462
	Summary	433	15-10	Matching donor and recipient for HLA class I and class II allotypes improves the outcome of organ transplantation	466
	Summary to Chapter 13	433	15-11	Allogeneic transplantation is made possible by the use of three types of immunosuppressive drug	466
	Questions	434	15-12	Corticosteroids change patterns of gene expression	467
	Chapter 14		15-13	Cytotoxic drugs kill proliferating cells	469
	Vaccination to Prevent Infectious Disease	437	15-14	Cyclosporin A, tacrolimus, and rapamycin selectively inhibit T-cell activation	470
14-1	Viral vaccines are made from whole viruses or viral components	437	15-15	Antibodies specific for T cells are used to prevent and control acute rejection	472
14-2	Bacterial vaccines are made from whole bacteria, their secreted toxins, or capsular polysaccharides	439	15-16	Patients needing a transplant outnumber the available organs	473
14-3	Adjuvants nonspecifically enhance the immune response	441			
14-4	Vaccination can inadvertently cause disease	443			

15-17	The need for HLA matching and immunosuppressive therapy varies with the organ transplanted	474		
	Summary	475		
	Transplantation of hematopoietic stem cells	476		
15-18	Bone marrow transplantation is a treatment for genetic diseases of blood cells	476		
15-19	Allogeneic bone marrow transplantation is the preferred treatment for many cancers	477		
15-20	The alloreaactions in bone marrow transplantation attack the patient, not the transplant	477		
15-21	Matching donor and recipient for HLA class I and II is particularly important in bone marrow transplantation	479		
15-22	HLA-identical bone marrow transplants cause GVHD through recognition of minor histocompatibility antigens	480		
15-23	Some GVHD helps engraftment and prevents relapse of malignant disease	481		
15-24	NK cells also mediate GVL effects	482		
15-25	Hematopoietic cell transplantation can induce tolerance to solid organ transplants	483		
	Summary	484		
	Summary to Chapter 15	484		
	Questions	485		
			Chapter 16	
			Cancer and Its Interactions with the Immune System	489
16-1	Cancer results from mutations that cause uncontrolled cell growth	490		
16-2	A cancer arises from a single cell that has accumulated multiple mutations	491		
16-3	Exposure to chemicals, radiation, and viruses can facilitate the progression to cancer	492		
16-4	Certain common features distinguish cancer cells from normal cells	494		
16-5	Immune responses to cancer have similarities with those to virus-infected cells	494		
16-6	Differences in MHC class I allow tumor cells to be attacked and eliminated by cytotoxic T cells	495		
16-7	Mutations in cellular genes acquired during oncogenesis provide tumor-specific antigens	496		
16-8	Cancer/testis antigens are a prominent type of tumor-associated antigen	498		
16-9	Successful tumors evade and manipulate the immune response	498		
16-10	By preventing infection, vaccination protects against cancers caused by viruses	500		
16-11	Vaccination with tumor antigens can cause cancer to regress	500		
16-12	Increasing co-stimulation can boost the T-cell response to tumor cells	501		
16-13	Heat-shock proteins can provide natural adjuvants of tumor immunity	501		
16-14	Monoclonal antibodies against cell-surface tumor antigens can be used for diagnosis and immunotherapy	502		
	Summary to Chapter 16	504		
	Questions	505		