Immunology: Autoimmune Disease Review Chart

	Symptoms	Type	Autoantigen	Disease Mechanism	Organ Specificity	Genetics	Gender (F:M)	Treatment Options	Tolerance Implications
Multiple Sclerosis (MS)	-Altered gait -Vision disturbances -Muscle weakness -Paralysis	-T-cell -Type IV	-MBP (myelin basic protein) -PLP (proteolipid proteins)	-CD4+ and T_H1 bind self-Ag causing demyelination of peripheral nerve axons -cytokine release -T_H2: oligoclonal bands of Ab in spinal cord	-Myelin sheath	HLA=DR2 RR=4.8 Strong	3:1 -Early reproductive years	-Anti-inflammatory drugs	-Failure of thymic deletion of self-reactive T-cells -Inflammatory environment reactivates anergic cells -Molecular/antigenic mimicry -Barrier break—release of sequestered Ag
Hashimoto's Thyroiditis	-Hypothyroidism	-T-cell	-Thyroid peroxidase -Thyroglobulin	-Infiltration of thyroid by cytoplasmic autoAg -Defect in negative feedback mechanism	-Thyroid	HLA=DR5 RR=3.2 Moderate	5:1		-Barrier break—release of sequestered Ag
Insulin-Dependent Diabetes Mellitus (Type I) (IDDM)	-Hyperglycemia -Kidney damage -HTN -Kidney infections -Glycosylated proteins on kidney	-Both T-cell and B-cell -Type IV	-Beta-cell in islets of Langerhans in pancreas -anti-ICA -anti-GAD	-CD4+, T_H1, CD8+, and CTL bind protein unique to beta cells -cytokine release -B-cells: ADCC	-Beta-cells in pancreas	HLA=DR3 and DR4 RR=14-25 Very strong	1:1 -Juvenile diabetes	-Insulin shots -Diet -Exercise -Beta-cell replacement therapy -Gene therapy -Secondary tissue damage	-Barrier break—release of sequestered Ag -Antigenic mimicry
Rheumatoid Arthritis (RA)	-Joint inflammation -Erosion of target cartilage -Lesions in blood vessel walls of synovial membrane -"Pannus" -Calcification w/in joints	-Both T-cell and B-cell -Type III and Type IV	-RF: rheumatoid factor – IgM anti-IgG -unknown synovial joint Ag	-CD4+, T_H1, CD8+, CTL, autoimmune B-cells direct tissue damage -RF production -abnormally hypoglycosylated IgG molecules -immune complex disease—fix complement -granulocyte deposition -lysosomal release, elastase, collagenase	-Joints -Synovial lining	HLA=DR4 RR=4-10 Strong	3:1	-Corticosteroids -Humanized Ab to TNF-alpha to lower inflammatory response -Highest placebo effect: 30%	-Cytokine imbalance w/ TNF-alpha -Barrier break—release of sequestered Ag -Molecular mimicry; cross reactions
Myasthenia Gravis	-Progressive muscle weakness -Flacid limbs -Diplopia (double vision) -Ptosis (droopy eyelids) -Slurred speech -Difficulty in chewing -Decreased muscle strength w/ repeated stimulation -Breathing problems	-Both T-cell and B-cell -Type II	-ACh receptor	-Competitive inhibitor of ACh receptor; antagonist -Fix complement -Transfer via placenta—IgG—transient disease	-ACh receptor at neuromuscular junction	HLA=DR3 RR=2.5-10 Very strong	1:1	-Thymectomy -Acetylcholinesterase inhibitors—pyridostigmine -Plasmapheresis to dilute [Ab]	

Immunology: Autoimmune Disease Review Chart, Continued

Goodpasture's Syndrome	-Inflammation -Cell damage—kidneys and lungs—hemorrhage	-B-cell -Type II	-Alpha 3 chain of Type IV collagen found in basement membranes of renal glomeruli and lung alveoli	-Ab bind Ag on basement membranes of kidney in "smooth" distribution	-Basement membranes of kidney	HLA=DR2 RR=13-16 Very strong	1:1	-Anti-inflammatory drugs -Kidney transplant	
Grave's Disease (Thyrotoxicosis)	-High T_3 and T_4 circulation -High metabolic activity -Sweating -Hot flashes -Nervousness -Weight loss -Goiter; thymus enlargement -Transient placenta transfer--IgG	-B-cell -Type II	-TSH receptor in thyroid (thyroid-stimulating hormone receptor)	-Overstimulation of thyroid (T_3, T_4) by auto-Ab that mimics effect of TSH by binding to TSH receptor; agonist	-Thyroid, TSH-receptor	HLA=DR3 RR=3.5 Moderate	5:1 peak incidence age 20-40	-Radioactive iodine treatment -Ablative surgery to remove thyroid, then treat w/ replacement of thyroid hormone	-Aberrant HLA Class II expression -Molecular mimicry; cross reactions with bacteria Yersinia enterolytica
Systemic Lupus Erythematosis (SLE)	-Glomerulonephritis -Vasculitis -Arthritis -Joint pain -Progressive disability -"Butterfly rash"—erythema w/ sunlight exposure	-B-cell; but T-cell dependent -Type III	-dsDNA -ssDNA -dsRNA -Histones, nuclear proteins -Ab to platelets, lymphocytes, RBCs	-Immune complex disease -"Lumpy-bumpy" distribution of Ab to basement membrane of kidney	-Multiple antigens; systemic effects	HLA=DR3 RR=6 Strong	15:1 (African-Americans/ Asian: 1/500 risk)	-NSAIDS -Immunosuppressive drugs -Plaquenil-anti malarial drug	-Enhanced T-cell helper function -Cytokine imbalance: AI "flare" w/ inflammmatory conditions -Molecular mimicry -Sequestered Ag -Polyclonal activation by superAg or LPS
Autoimmune Hemolytic Anemia (AHA)	-Anemia -High complement activation-RBCs	-B-cell -Type II	-Rh blood group antigens	-RBC binds directly to Abs (IgG, IgM) to target destruction by complement (classical) of tagged RBCs	-RBC—Rh blood group antigens				
Pernicious Anemia	-Absence of vitamin B_{12} in RBCs -Low # of RBCs -Deficient RBC development; erythropoiesis	-B-cell	-IF; intrinsic factor; vit.B_{12} transporter	-Auto-Ab binds to IF preventing B_{12} transport across intestinal mucosa; thus deficient RBC development	-IF, but systemic effects; deficient RBC develop; erythropoiesis	-Increased incidence of second autoimmune disease--autoimmune thyroiditis			
Autoimmune Thrombocytopenia Purpura (ITP)	-Abnormal bleeding	-B-cell -Type II	-Platelet integrin	-Ab against cell surface or matrix Ag	-Platelets, but systemic effects				

*__Note:__ Type II, III, and IV—correspond to types of Hypersensitivity Reactions (varies by textbook).
RR = relative risk; shows how likely HLA haplotype is linked with autoimmune susceptibility.