IMMUNOPATHOLOGY REVIEW 2016					
Immunopathology	**Mechanism**	**Effector cells**	**Typical example**	**Diagnosis**	**Treatment**
Type 1	IgE on mast cells ►Early phase: histamine release (15 min) ►Late phase: PG and LT production, cytokines (6-8 hr.) ►Eosinophil chemotaxis	(B cells make IgE) (excessive Th2-like Tfh help) Mast cells Various, including M2 macrophages Eosinophils	►Seasonal rhinitis (mostly early phase) ►Asthma (eventually mostly late phase) ►Eczema (chronic late phase) ►Anaphylaxis (early phase)	-Skin prick tests -History -CAP-FIA (lab test for specific IgE; similar to an ELISA) -Challenge tests	►Antihistamines ►Corticosteroids ►Leukotriene antagonists ►mAb to IgE (omalizumab, Xolair) for chronic asthma ►Epinephrine for anaphylaxis
	The other half of allergy/helminth immunology	Th2 cells attract M2 macrophages and eosinophils	Worm killing or encapsulation; scarring in asthma	Biopsy and immunohistochemistry	►Corticosteroids
Type 2	IgG autoAb (occasionally IgA, IgM)	(B cells make IgG) (excessive or hybrid self-foreign Tfh help; cross-reaction; exposure of sequestered Ag) Neutrophils	►Graves disease ('LATS' Ab to TSH receptor *stimulates*) ►Goodpasture (Ab to collagen Ag in kidney and lung) ►Rheumatic heart disease (cross-reaction with Strep)	-Fluorescent antibody techniques using patient's or normal tissue (linear pattern) -Some specific immunoassays	►Corticosteroids ►NSAIDs (nonsteroidal anti-inflammatory drugs) ►DMARDs (disease-modifying anti-rheumatic drugs) ►mAbs to decrease T or B cells
Type 3	IgG, IgA (IgM) immune complexes to intrinsic or extrinsic Ags get trapped in basement membranes	(B cells make Ab) (excessive or illicit Tfh help) Neutrophils	►Serum sickness ►Poststreptococcal glomerulonephritis ►Hypersensitivity pneumonitis (Farmer's Lung: develops into a Type 4 disease) ►Arthus reaction	-Rheumatoid factor -Biopsy shows lumpy-bumpy pattern	-Supportive care for self-limited conditions -Corticosteroids

Type 4	T cell mediated (delayed hypersensitivity)	Th1, Th2, Th17 (insufficient Treg) M1 macs (Th1) M2 macs (Th2) CTL	►Multiple sclerosis ►Hashimoto thyroiditis ►Type 1 diabetes (T1D) ►Rheumatoid arthritis (like T1D, starts with autoAb then → T cell dominated	-Biopsy shows mononuclear cell infiltrate -Research tests show Ag-specific T cells	-Immunosuppress -Anti-inflammatory -mAbs to T cells -sometimes, mAbs to B cells (MS, RA)
'Chronic Frustrated Immune Response'	T cells against non-removable Ags → chronic inflammation	Th1, Th2, Th17 (insufficient Treg) M1 macs (Th1) M2 macs (Th2) CTL	►celiac disease: gluten ►inflammatory bowel disease (Crohn, ulcerative colitis): normal gut flora ►Chronic beryllium disease: Inhaled Be dust ►psoriasis: normal skin flora	-Biopsy shows characteristic pathology. -Research tests show Ag-specific T cells, insufficient Treg.	-Immunosuppress -Anti-inflammatory -mAbs to T cells

Strongest HLA associations (every immune disease has *some* HLA linkage)	
Rheumatoid arthritis	HLA-DR4, HLA-DR1
Type 1 diabetes	HLA-DR3, HLA-DR4 in linkage disequilibrium with the real culprits, HLA-DQ2 and HLA-DQ8 HLA-DR2 is protective
Ankylosing spondylitis	HLA-B27
Systemic lupus erythematosus	HLA-DR3
Celiac disease	HLA-DQ2, -DQ8