<table>
<thead>
<tr>
<th>Immunology: Autoimmune Disease Review Chart</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Symptoms</strong></td>
</tr>
<tr>
<td>--------------------------------------------</td>
</tr>
</tbody>
</table>
| **Multiple Sclerosis (MS)** | - Altered gait  
- Vision disturbances  
- Muscle weakness  
- Paralysis | - T-cell  
- Type IV | - MBP (myelin basic protein)  
- PLP (proteolipid proteins) | - CD4+ and Tα1 bind self-Ag causing demyelination of peripheral nerve axons  
- Cytokine release  
- TII2: oligoclonal bands of Ab in spinal cord | - Myelin sheath | 3:1 | Early reproductive | - 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 | 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+, Tα1, CD8+, and CTL bind protein unique to beta cells  
- Cytokine release  
- B-cells: ADCC | - Beta-cells in pancreas | 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+, Tα1, CD8+, and CTL, autoimmune B-cells direct tissue damage  
- RF production  
- Abnormally glycosylated IgG molecules  
- Immune complex disease — fix complement  
- Granulocyte deposition  
- Lysosomal release, elastase, collagenase | - Joints  
- Synovial lining | 3:1 | - Corticosteroids  
- Humanized Ab to TNF-alpha to lower inflammatory response  
- Highest placebo effect: 30%  
- Barrier break — release of sequestered Ag  
- Molecular mimicry; cross reactions |
| **Myasthenia Gravis** | - Progressive muscle weakness  
- Flaccid 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 | 1:1 | - Thymectomy  
- Acetylcholinesterase inhibitors — pyridostigmine  
- Plasmapheresis to dilute [Ab] |
| | | | | | | | | |
## Immunology: Autoimmune Disease Review Chart, Continued

<table>
<thead>
<tr>
<th>Goodpasture’s Syndrome</th>
<th>-Inflammation</th>
<th>-Cell damage—kidneys and lungs—hemorrhage</th>
<th>-B-cell</th>
<th>-Type II</th>
<th>-Alpha 3 chain of Type IV collagen found in basement membranes of renal glomeruli and lung alveoli</th>
<th>-Ab bind Ag on basement membranes of kidney in “smooth” distribution</th>
<th>-Basement membranes of kidney</th>
<th>HLA=DR2 RR=13-16 Very strong</th>
<th>1:1</th>
<th>-Anti-inflammatory drugs</th>
<th>-Kidney transplant</th>
</tr>
</thead>
<tbody>
<tr>
<td>Grave’s Disease (Thyrotoxicosis)</td>
<td>-High T3 and T4 circulation</td>
<td>-High metabolic activity</td>
<td>-Sweating</td>
<td>-Hot flashes</td>
<td>-Nervousness</td>
<td>-Weight loss</td>
<td>-Goiter; thymus enlargement</td>
<td>-Transient placenta transfusion</td>
<td>-IgG</td>
<td>-B-cell</td>
<td>-Type II</td>
</tr>
<tr>
<td>-Molecular mimicry; cross reactions with bacteria Yersinia enterolytica</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Systemic Lupus Erythematosus (SLE)</td>
<td>-Glomerulonephritis</td>
<td>-Vasculitis</td>
<td>-Arthritis</td>
<td>-Joint pain</td>
<td>-Progressive disability</td>
<td>-“Butterfly rash”—erythema w/ sunlight exposure</td>
<td>-B-cell; but T-cell dependent</td>
<td>-Type III</td>
<td>-dsDNA</td>
<td>-ssDNA</td>
<td>-dsRNA</td>
</tr>
<tr>
<td>-Enhanced T-cell helper function</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>-Cytokine imbalance: AI “flare” w/ inflammatory conditions</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>-Molecular mimicry; Sequestered Ag</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>-Polyclonal activation by superAg or LPS</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Autoimmune Hemolytic Anemia (AHA)</td>
<td>-Anemia</td>
<td>-High complement activation-RBCs</td>
<td>-B-cell</td>
<td>-Type II</td>
<td>-Rh blood group antigens</td>
<td>-RBC binds directly to Abs (IgG, IgM) to target destruction by complement (classical) of tagged RBCs</td>
<td>-RBC—Rh blood group antigens</td>
<td>-IF, intrinsic factor; vit.B12 transporter</td>
<td>-Auto-Ab binds to IF preventing B12 transport across intestinal mucosa; thus deficient RBC development</td>
<td>-IF, but systemic effects; deficient RBC develop; erythropoiesis</td>
<td>-Increased incidence of second autoimmune disease—autoimmune thyroiditis</td>
</tr>
<tr>
<td>Pernicious Anemia</td>
<td>-Absence of vitamin B12 in RBCs</td>
<td>-Low # of RBCs</td>
<td>-Deficient RBC development; erythropoiesis</td>
<td>-B-cell</td>
<td>-IF</td>
<td>-Auto-Ab binds to IF preventing B12 transport across intestinal mucosa; thus deficient RBC development</td>
<td>-IF, but systemic effects; deficient RBC develop; erythropoiesis</td>
<td>-Increased incidence of second autoimmune disease—autoimmune thyroiditis</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Autoimmune Thrombocytopenia Purpura (ITP)</td>
<td>-Abnormal bleeding</td>
<td>-Platelet integrin</td>
<td>-Ab against cell surface or matrix Ag</td>
<td>-Platelets, but systemic effects</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*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.