Please note that the Bioscience II web page has been updated with the following additions:

1) Answers to quiz 6
2) A practice exam for exam 5
3) Learning objectives for exam 5
4) Lecture notes containing revisions to 3/15 and 3/17 lectures (revised notes for 3/19 will be added today)

Prophylaxis – protection from disease, as is provided by antisera

Anaphylaxis – the development of IgE-mediated hypersensitivity to relatively harmless substances

Hypersensitivities = allergies; immune responses that cause tissue damage

Sensitized – previous exposure to antigen creating an allergy

Autoimmune disease - inappropriate response to self antigens

Immunodeficiency - ineffective immune system; inadequate response
Topics

- Type I hypersensitivity
- Type II hypersensitivity

Type I hypersensitivity
Type I hypersensitivity

- Immediate IgE–mediated
- Localized anaphylaxis
- General anaphylaxis
- Immunotherapy

Figure 18.1 - Immediate IgE–mediated
**Figure 18.2 Localized anaphylaxis**

*Hives* – allergic skin reaction characterized by formation of a wheal and flare blocked by antihistamines.

*Hay fever* – antigen is inhaled, causing localized anaphylaxis in tissues below mucous membranes blocked by antihistamines.

*Asthma* – localized anaphylaxis causes increased mucous secretion, bronchial spasms non-histamine mediators primarily responsible; antihistamines not effective albuterol – bronchodilator steroids – inhibit inflammatory reaction
Generalized anaphylaxis

- Antigens become widespread via bloodstream
- Shock (reduced blood pressure) – loss of fluid from blood vessels into tissues
Generalized “systemic” anaphylaxis

- Antigens become widespread
- Shock (reduced blood pressure)
- Ex. Bee stings, peanuts and penicillins
  
  *penicillin converted to hapten-protein complex*
  
  *complex elicits IgE antibodies*

*Controlled by epinephrine (adrenalin)*

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**Figure 18.3**

Immunotherapy

*Desensitization, hyposensitization therapy*
Type II hypersensitivity

- Cytotoxic
- Transfusion reactions
  - Hemolytic diseases

Cytotoxic

- Complement lysis
- Antibody – dependent cellular cytoxicity (ADCC)
### Table 18.2 - Antigens and Antibodies in Human ABO Blood Groups

<table>
<thead>
<tr>
<th>Blood Type</th>
<th>Antigen Present on Erythrocyte Membranes</th>
<th>Antibody in Plasma</th>
<th>Incidence of Type in United States</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>Among Whites</td>
</tr>
<tr>
<td>A</td>
<td>A</td>
<td>Anti-B</td>
<td>41%</td>
</tr>
<tr>
<td>B</td>
<td>B</td>
<td>Anti-A</td>
<td>19%</td>
</tr>
<tr>
<td>AB</td>
<td>A and B</td>
<td>Neither anti-A nor anti-B</td>
<td>4%</td>
</tr>
<tr>
<td>O</td>
<td>Neither</td>
<td>Anti-A and anti-B</td>
<td>45%</td>
</tr>
</tbody>
</table>

IgM antibodies cause a Type II hypersensitivity reaction
Foreign erythrocytes are agglutinated by recipients antibodies
Complement is activated
Red blood cells are lysed
Why is it surprising that people lacking the A or B antigen are found to have antibodies to the corresponding antigen?

Figure 18.4 Hemolytic disease

Small amount of fetal Rh antigen induces secondary response; Mother’s IgG crosses placenta

Rh antigen may also be introduced via induced or spontaneous abortion
Why is Rh-negative blood used to transfuse the fetus or newborn?

Why do Rh-negative but not Rh-positive mothers sometimes have babies with hemolytic disease of the newborn?