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

Figure 18.1 - Immediate IgE – mediated

Cytokines induce IgE producing B cells in tissues under mucous membranes; more abundant in allergic individuals
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

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)

Figure 18.3

Immunotherapy

Desensitization, hypo sensitization therapy
Type II hypersensitivity

- Cytotoxic
- Transfusion reactions
- Hemolytic diseases

Cytotoxic

- Complement lysis
- Antibody–dependent cellular cytotoxicity (ADCC)

**Table 18.2 - Transfusion reactions**

<table>
<thead>
<tr>
<th>Blood Type</th>
<th>Antigen Present on Erythrocytes</th>
<th>Antibody in Plasma</th>
<th>Incidence of Type II in United States</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>A</td>
<td>Anti-A</td>
<td>63% Among Whites 23% Among Asians 21% Among Blacks</td>
</tr>
<tr>
<td>B</td>
<td>B</td>
<td>Anti-B</td>
<td>12% Among Whites 27% Among Asians 20% Among Blacks</td>
</tr>
<tr>
<td>AB</td>
<td>A and B</td>
<td>Anti-A and anti-B</td>
<td>4% Among Whites 5% Among Asians 7% Among Blacks</td>
</tr>
<tr>
<td>O</td>
<td>Neither</td>
<td>Anti-A and anti-B</td>
<td>40% Among Whites 40% Among Asians 40% Among Blacks</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?