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

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Generalized anaphylaxis

- Antigens become widespread via bloodstream
Generalized anaphylaxis

- Antigens become widespread
- 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)

Figure 18.3
Immunotherapy

Desensitization, hyposensitization therapy
Type II hypersensitivity

- Cytotoxic
- Transfusion reactions
- Hemolytic diseases

Cytotoxic

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

Table 18.2 - Transfusion reactions

IgM antibodies cause a Type II hypersensitivity reaction
Foreign erythrocytes are agglutinated by recipient's 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?

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

Figure 18.4 Hemolytic disease
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?