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)*

18-1

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

18-2

Topics

- **Type I hypersensitivity**
- **Type II hypersensitivity**

18-3

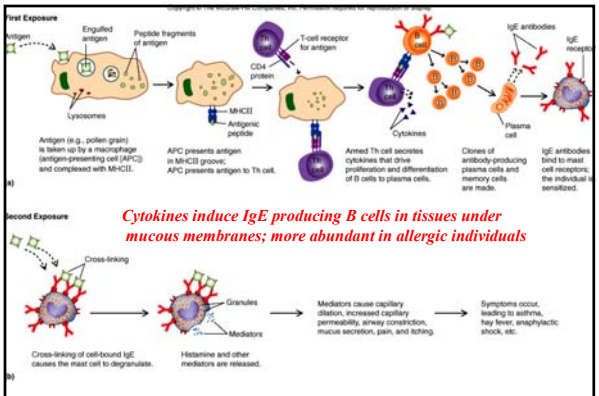
Type I hypersensitivity

18-4

Type I hypersensitivity

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

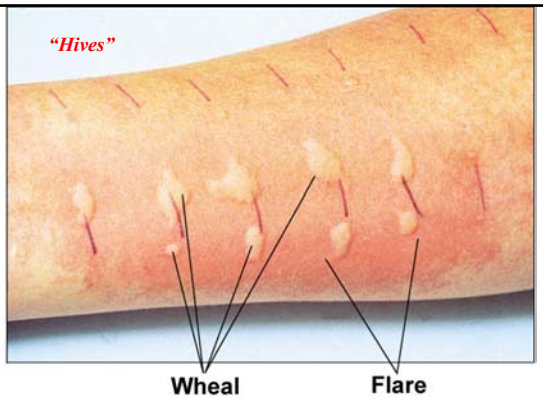
18-5



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

18-6

Figure 18.1 - Immediate IgE – mediated



18-7

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

18-8

Generalized anaphylaxis

- Antigens become widespread *via bloodstream*

18-9

Generalized anaphylaxis

- Antigens become widespread
- Shock (reduced blood pressure) – *loss of fluid from blood vessels into tissues*

18-10

Generalized "*systemic*" anaphylaxis

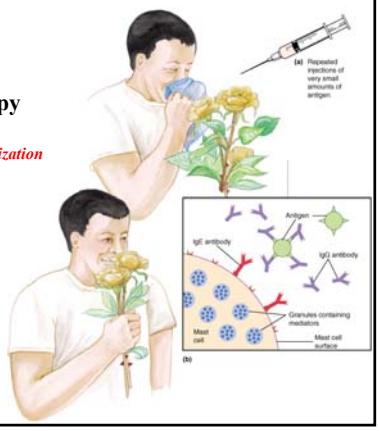
- Antigens become widespread
- Shock (reduced blood pressure
- Ex. Bee stings, peanuts and penicillins
  penicillin converted to hapten-protein complex
  complex elicits IgE antibodies

Controlled by epinephrine (adrenalin)

18-11

Figure 18.3
Immunotherapy

Desensitization, hyposensitization therapy



18-12

Type II hypersensitivity

- Cytotoxic
- Transfusion reactions
- Hemolytic diseases

18-13

Cytotoxic

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

18-14

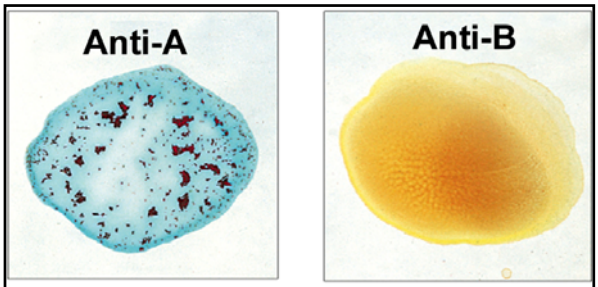
TABLE 18.2 Antigens and Antibodies in Human ABO Blood Groups

Blood Type	Antigen Present on Erythrocyte Membranes	Antibody in Plasma	Incidence of Type in United States		
			Among Whites	Among Asians	Among Blacks
A	A	Anti-B	41%	28%	27%
B	B	Anti-A	10%	27%	20%
AB	A and B	Neither anti-A nor anti-B	4%	5%	7%
O	Neither	Anti-A and anti-B	45%	40%	46%

Table 18.2 - Transfusion reactions

IgM antibodies cause a Type II hypersensitivity reaction
Foreign erythrocytes are agglutinated by recipients antibodies
complement is activated
red blood cells are lysed

18-15

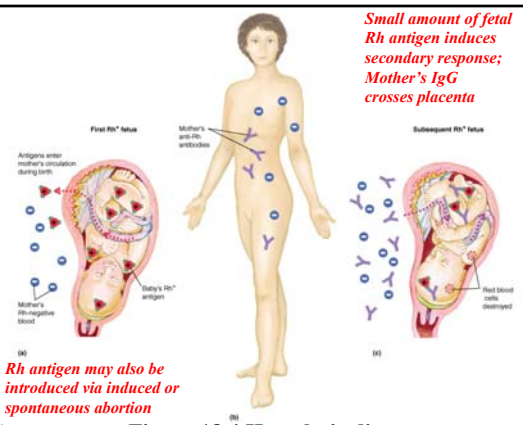


18-16

Why is it surprising that people lacking the A or B antigen are found to have antibodies to the corresponding antigen?

18-17

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

18-18

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?

18-19