BIO 226N
STUDY GUIDE
IMMUNOLOGY LECTURES

SPECIFIC RESISTANCE

A. HUMORAL IMMUNITY

Antigens - provoke AB synthesis
Properties - foreign
- high molecular weight ≥ 10,000
- degradable by host

Examples - proteins on bacteria, viruses
- pollens, dust, dander, egg white
- transplanted tissue/organs

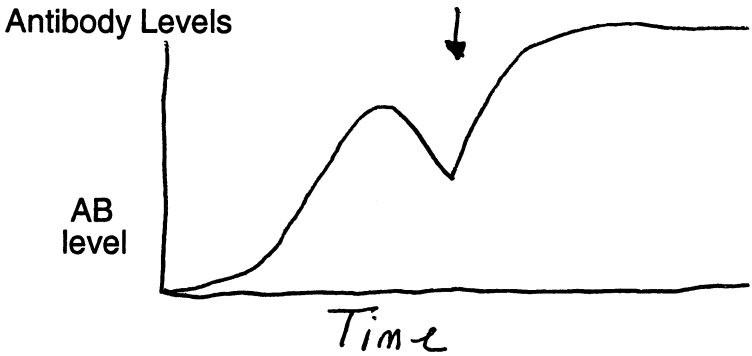
Antigenic Determinants

Antibodies = gamma-globulins = immunoglobulins = a certain class of serum proteins
[synthesized & secreted by some lymphocyte derivatives]
- plasma (circulate)
- bind to AG, help destroy
- specific; binding sites
- 2 heavy, 2 light chains
- constant and variable regions

Antibody Synthesis
gene → mRNA → translation
lymphocytes (T & B)
stem cells in bone marrow or liver T & B
B lymphocytes synthesize AB

STEPS OF ANTIBODY SYNTHESIS AFTER INJECTION OF T-DEPENDENT ANTIGEN

1. Macrophages ingest, digest, display antigenic determinants on macrophage surface
2. Now called antigen presenting cells (APC)
3. APC have self markers also on surface
4. APC + T helper cell binds
5. APC + T helper & B cell (pre-existing which can synthesize AB to that the AG)
6. Those B cells - stimulated to grow & divide and mature into plasma cells which produce and secrete AB
7. A few of these B cells become memory cells



Immunity to: Bacteria - *Bordetella pertussis*
Sal. typhi
Exotoxins - *Clostridium tetani* toxin
C. diphtheriae toxin
Viruses - Polio, Common cold, Hepatitis B, Influenza

B. CELL-MEDIATED IMMUNITY (CMI)

I. CMI involves T-lymphocytes
   a. Receptors
   b. React with foreign antigens on the surface of our own cells such as viruses budding through cytoplasmic membrane

II. Stem cells in the bone marow become many different kinds of cells
   a. Neutrophils, basophils, eosinophils, monocytes, etc.
   b. Some develop into Pre-B-lymphocytes
   c. Some migrate to thymus and become immature T-lymphocytes (T-cells)

III. T cells can react with a huge variety of antigens
   a. Surface proteins (receptors) that resemble immunoglobulins
   b. Antigen recognized on APC
   c. Self markers also on APC

IV. Antigen-stimulated T cells mature and divide (proliferate) and become:
   a. Cytotoxic T cells (T_C)
   b. Helper T-cells (T_H)
   c. Suppressor T-cells (T_S)
   d. Delayed type hypersensitivity (T_D)

Natural Killer Cells not really either T or B
Killer Cells

V. Cellular immunity combats:
   a. Intracellular viruses
   b. Multicellular parasites
   c. Cancer
   d. Some bacteria (Mycobacterium, Rikettsia)
   e. Transplanted tissues

C. DUALITY OF THE IMMUNE SYSTEM

I. Immune deficiencies
   a. Aggammaglobulinemia -- reduced (or no) circulating antibodies
   b. DiGeorge syndrome--no thymus & no CMI No T_C Lymphocytes

II. Both types of immunity of essential for health

D. VACCINES

I. Stimulate production of specific Antibodies or specific cytotoxic T-cells (T_C).

II. Bacterial vaccines
   a. *Bordetella pertussis*, a killed vaccine
   b. *Mycobacterium tuberculosis* strain BCG, an attenuated vaccine

III. Viral vaccines
   a. Polio
      1. First killed (Salk), then attenuated
      2. Grown in tissue cultures - monkey kidney cells
   b. Rabies -- killed or attenuated
   c. Smallpox
   d. Live, attenuated virus vaccines usually give better immunity than inactivated viruses

IV. Toxins/Toxoids
   a. Toxins often cause disease symptoms
   b. Antibodies against a toxin can neutralize it and prevent disease
      1. Toxoid = altered toxin
      2. DPT vaccine

V. Subunit Vaccines (Hepatitis B)

VI. Antiserum
   a. Pooled normal human serum
   b. Human with known antibody
   c. Purified human gamma globulin
   d. Serum from immunized animal

E. SEROLOGY

I. The study or use of antigen-antibody reactions in the laboratory

II. There are many types of antigen-antibody reactions and many ways to detect them
   a. Agglutination
   b. Hemagglutination
   c. Precipitation
   d. Toxin or virus neutralization

F. ACQUISITION OF IMMUNITY

I. Active immunity: body makes antibodies and/or specific T_C
   a. Natural -- infection and recovery with Ab production
   b. Artificial -- vaccination

II. Passive Immunity
   a. Natural -- fetus receives maternal antibodies while *in utero*
   b. Artificial -- injection of antiserum

G. IMMUNE DISORDERS OR HYPER-SENSITIVITIES (allergy) humoral or CMI: immediate or delayed

I. Anaphylaxis - humoral - IgE
   immune IgE binds basophils and mast cells surfaces and coats them; AG (e.g. pollen) bridges to adjacent IgE
   The cells release granules, includes mediators (histamine)
   Mediators cause inflammation, mucous secretion, smooth muscle contraction, breathing difficulty
   a. localized -
      digestive tract (food) vomit, diarrhea
      respiratory tract (pollen, house dust, fungal spores, dander)
      upper - itchy, watery eyes, cough, sneeze = hay fever
      lower - smooth muscle contraction, asthma
      Adrenalin = Epinephrine
   b. Systemic - Generalized
      Bee sting, penicillin 2%
      itch, rash, faint, dilation of blood vessels, blood pressure, drop, shock, death
      Adrenalin
      Desensitization

II. CYTOTOXIC REACTIONS
IgG or IgM react - AG on host blood or other tissue cell - lysis

a. transfusion reactions
ABO blood groups
AG, AB, genes
fever, prostration, kidney failure, shock, death

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determining blood type, donor cells & known serum
anti A or anti B agglutination with

known serum	group of donor
anti A	A
anti B	B
anti A & anti B	AB
no agglutination with anti A nor anti B	O

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cross match (donor and recipient) to make sure there is no agglutination
major
donor RBC & recipient serum
minor
donor serum & recipient RBC
universal donor = O blood group
universal recipient = AB group, have no anti A or anti B

b. HEMOLYTIC DISEASE OF NEWBORN- RHESUS FACTOR
People 85% Rh+ and 15 % Rh-
Rh+ Father+Rh- Mother → Rh+ Child
Rh+ RBC from Fetus enter Mother, cause antibody ynthesis
subsequent pregnancy with Rh+Fetus
anti Rh antibody cross placenta; enter Fetus

anti Rh antibody & Rh+ RBC of Fetus → RBC Destruction

RESULT: Decrease in O_2 transport & increase in bilirubin level

AT BIRTH: Bilirubin cannot be metabolized by newborn baby's liver

TREATMENT
i. monitor expectant mother anti Rh
ii. fluorescent light on child
iii. monitor newborn bilirubin level
iv. blood exchange with Rh- blood after birth
v. passive immunize expectant mother
vi. infusion in utero in extreme cases

III. IMMUNE COMPLEX REACTIONS
Small Antigen-antibody complexes escape phagocytosis
Complexes deposited in tissues, cause inflammation
Phagocytes release digestive enzymes which damage host
a. Acute Post-Streptococcal Glomerulonephritis-inflammation of glomeruli in kidneys
b. Rheumatoid Arthritis - complexes in joints
c. Systemic lupus erythematosis - Antibodies to own nucleic acid

IV. DELAYED HYPERSENSITIVITY -CMI-T lymphocytes

24-48 hrs
contact dermatitis (poison ivy, cosmetics, metal)
tuberculin hypersensitivity
granulomatous hypersensitivity

H. TOLERANCE/AUTOIMMUNITY
body does not (normally) make AB to itself
sometimes we do
rheumatic fever - antistreptococcal AB react with heart valve

I. TRANSPLANTATION
major antigens on tissues differ in different individuals
tissue rejection

J. IMMUNOSUPPRESSION -
cyclosporin - suppresses CMI
heart or kidney transplant patients

K. IMMUNE DEFICIENCIES - SUMMARY
1. INHERITED
a. Hypogammaglobulinemia
b. Agammaglobulinemia
c. DiGeorge Syndrome

2. ACQUIRED – HIV/AIDS

metabolized by newborn baby's liver